THE
LARYNGOSCOPE.

VOL. XIX. ST. LOUIS, MO., NOVEMBER, 1909. No. 11.

ORIGINAL COMMUNICATIONS.

(Original Communications are received with the understanding that they are contributed exclusively to THE LARYNGOSCOPE.)

THE OCULO-ORBITAL, INTRACRANIAL AND CEREBRAL COMPLICATIONS OF DISEASES OF THE NASAL ACCESSORY SINUSES.

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In the etiology of oculo-orbital, intracranial and cerebral complications of the accessory sinuses, infection and extension of inflammatory processes must be considered as important factors. Infection may extend through the tissues as readily as by way of the vascular system. The especial carriers of infection are the streptococci and staphylococci, and the rapidity of extension depends on their amount of virulence. The avenue of infection depends on the continuity and vascularity of the mucosa of the accessory sinuses. The lesions of the accessory sinuses, the virulence of the source of infection, the thrombi in the venous channels of the mucosa of the accessory sinuses indicate the co-existent attack on the bony walls and direct contact-infection of the cranium and of the contents of the orbital cavity. Thrombo-phlebitis, detachment of the thrombus and the bacteria found in the blood vessels lead to the extension of the process, as indicated by local and general symptoms. Thrombosis occurring in the veins in the mucosa of the accessory sinuses may extend through the bone to the dura mater and thence to the cranial sinuses. The frequent involvement of the thin bony walls of the accessory sinuses may lead to adhesions between the inner sinus walls and the meninges or the brain, or even to new vascular anastomosis.

*Presented, by invitation, to the American Academy of Ophthalmology and Oto-Laryngology, October 4, 1909.

The oculo-orbital, intracranial and cerebral complications of the diseases of the accessory sinuses may be caused by direct infection or by tissue continuity; this direct infection may be greatly intensified by congenital or acquired bone dehiscence. The diseased mucosa of the accessory sinuses may come in direct contact with the periorbita, the dura mater or the sheath of the optic nerve, and in this way the morbid process may extend to these specified parts. The dehiscence of the cribriform plate of the ethmoid may produce the communication with the orbita, those of the canalis opticus with the optic nerve. Complications may also be directly caused by disease of the venous anastomoses of the mucosa of the accessory sinuses and of the meninges or sinuses, or furthermore of the opthalmic veins and by the bacteria which have found their way into these channels. The indirect oculo-orbital, intracranial and cerebral complications may be produced by secondary affection of the accessory sinuses adjacent to the cranial and orbital cavities, or by affection of the more remote vessels and venous anastomoses; furthermore, the indirect intracranial and cerebral complications may result from involvement of the orbital contents through the walls of the orbit, the motor nerves of the eye and the optic nerve. The lymph vessels also participate both in the direct and indirect infection of the contents of the cranial and orbital cavities.

In the direct infection of the cranial and orbital cavities we must consider the size of the accessory sinuses, the extension of their cerebral walls in the anterior, middle and posterior cranial fossae, and of their orbital walls to the orbit; furthermore, the strength and thickness of these bony walls, their dehiscences, their emissary vessels, the mucosa of the accessory sinuses, the diploe, the meninges and sinuses of the cranium must be considered. It is self-evident that the size of the accessory sinuses exerts an etiologically favorable influence in the production of their diseases and complications.

If we assume from occasional anatomical data and from statistics concerning the frontal sinus, as Gerber has done, that the left frontal sinus is larger and therefore more easily diseased, then the anatomical data must be excluded, for the anatomic expansion of the left frontal sinus and its more frequent disease must be considered only as a circumstance. Our radiographs of the frontal sinuses of 1,200 skulls show in horizontal and vertical axes as follows: In one-third of the frontal sinuses both sides appear approximately equally developed; in one-third the right sinus was larger; in one-third the left sinus was larger.

The greater number of venous anastomoses between the accessory sinus mucosa and the meningeal and orbital venous network depends largely on the more extensive development of the individual sinuses. As also does the larger contact-surface of the individual sinuses with the dura, the cranial fossae and the periosteum of the orbit. These facts are important etiological factors in the oculo-orbital, intracranial and cerebral complications. The pathological changes in the cerebral and orbital sinus walls, as shown histologically and microscopically, prove the direct infection of the cranial and orbital contents in a large percentage of cases. Perforations of the individual sinuses into the anterior and middle fossae and also into the orbit have been frequently observed.

Especially associated with involvement of the cerebral walls of the accessory cavities is inflammation of the adjacent areas of the dura mater; in this manner pachymeningitis externa, extra-dural abscess, pachymeningitis interna and intra-dural abscess may ensue; thus, also, involvement of the orbital accessory sinus walls may be followed by inflammation of the adjoining areas of the periorbita, producing orbital periostitis, orbital abscess and orbital phlegmon. Leptomeningitis, thrombo-phlebitis and brain abscess may be developed as a further extension of such a process or by direct extension. Here, too, the veins of the accessory sinuses, the veins of the diploe, and the direct and indirect venous anastomoses with the dural and meningeal venous network, are factors.

We will first consider the cerebral and orbital walls of the individual accessory cavities. The frontal sinus usually extends largely into the squamous part of the frontal bone, which frequently corresponds to the area of the gyrus frontalis medius; at the border of the horizontal plate of the frontal bone the frontal sinus encroaches on the area of the sulcus fronta marginalis, the junction of the convex surface with the lower surface of the frontal lobe; the latter may also encroach on the superior wall of the orbit, depending on the development of the frontal sinus. Where entreme enlargement is found, the frontal sinus may extend widely to the entire anterior cranial fossa and to the entire upper wall of the orbit; in one case which I have observed the frontal sinus extended to the middle cranial fossa between the fissura orbitalis superior and the temporal fossa. In such cases the frontal sinus may be brought into close relationship with the optic nerve, which emerges through the foramen opticum, to the eye nerves, emerging from the fissura orbitalis superior, to the under surface of the frontal lobe and to the anterior part of the temporal lobe. The frontal sinus

may extend superiorly, according to our observations, from 4 to 7.5 cm. to the region of the gyrus frontalis superior and medius. The frontal sinus may extend assymmetrically to the area of the opposite frontal lobe; this extension into the area of the opposite lobe varied in our cases from 10 to 35 mm. in width.

In addition to these enumerated etiological factors, an abscess may form on the convex surface and base of the frontal lobe, due to disease of the cerebral wall of the frontal sinus and its perforation caused by involvement of the cerebral membranes; abscess of the temporal lobe and of the opposite frontal lobe may also be developed. The enumerated assymmetries of the frontal sinuses explain the morphological basis of origin of contra-lateral and bilateral cerebral abscesses in an unilateral frontal sinus affection. Disease of the orbital frontal sinus wall and its subsequent perforation may produce complications of the tear-ducts and the eyeball and orbital phlegmon with its sequellae; these assymmetries of the frontal sinus may also explain the origin of contra-lateral orbital complications.

The ethmoid cells form the cross-section of the ethmoid labyrinth corresponding medially to a part of the anterior cranial fossa and laterally to a point of the inner wall of the orbit. The anterior orbital cells may extend into the anterior cranial fossa. In our cases, the width of the anterior orbital cells varied from 10 to 29 mm.; the width of the posterior orbital cells varied from 12 to 21 mm.; with these extensions they may encroach upon the lower surface of the frontal lobe, and on the superior wall of the orbit. The posterior or sphenoid ethmoid cells extend into the area of the sphenoid bone. In our cases they varied in measurement from 8 to 40 mm. These posterior cells may form part of the superior orbital walls, and half of the sella turcia, and may extend to the fissura orbitalis superior and to the foramen rotundum. The posterior ethmoid cells may, according to our observations, be brought into closer relationship on both, or only on one side, with the single walls of the canalis opticus and its single segments of the pulcus opticus; furthermore, both the right and the left posterior ethmoid cells encroach on the canalis opticus and the sulcus opticus, and consequently may encroach on the anterior cranial fossa of the opposite side.

The ethmoid cells encroach most frequently on the posterior surface of the frontal lobe to the region of the gyrus rectus and to a part of the gyrus orbitalis. The fronto-orbital cells may, in some cases, touch the entire under surface of the frontal lobe. The posterior ethmoidal cells may medially reach to the area of the tuber-cinereum and laterally to the temporal lobe. In unusual cases

the posterior ethmoid cells may extend to the area of the lower surface of the frontal lobe of the opposite side.

In addition to the enumerated etiological factors, an abscess in the frontal lobe or in the temporal lobe of the same side may result from disease of the cerebral walls of the ethmoid cells and perforation of same, with complications of the cerebral membranes; furthermore, involvement of the opposite frontal lobe may ensue without involvement of the chiasma or either optic nerve. The diseases of the contents of the orbit and their sequeliae may arise from disease of the orbital walls of the ethmoid cells.

The sphenoid cavity has two cerebral walls, namely, (a) superior and (b) lateral wall; these form a part of the middle cerebral fossa. Furthermore, the sphenoid cavity may form a part of the superior and inner wall of the orbit in the posterior part of the orbital cavity. Usually the superior wall of the sphenoid cavity forms the sella turcica and bounds the hypophysus; the lateral sphenoidal wall, together with the sulcus caroticus, comes in contact with the carotis interna and the sinus cavernosus. The sella turcica may at best be formed only partly of the wall of the sphenoid cavity. It may extend asymmetrically, according to our observations, to the anterior and middle cranial fossa of the opposite side; it may also extend along the median line above the nasal septum 1.5 cm., anterior to the osteum sphenoidale, and may thus touch both anterior cranial fossae medially. The longest cross-diameter of the sphenoid cavity which we observed was 6 cm. The sphenoidal cavity may be in close relationship with one wall of the canalis opticus and with single segments of the sulcus opticus of either one or both sides; likewise we have observed that both the right and left sphenoidal cavities may bound the canalis opticus and sulcus opticus of the opposite side.

The sphenoidal cavity may also border on the posterior cranial fossa in varying dimensions; in one case we observed the sphenoid cell 10 mm. from the dorsum sellae, and extending 30 mm. to the clivus. The sphenoid cavity lies usually in the area of the tuba cinereum and the temporal lobe. It may border on the lower surface of the frontal lobe of both the same and the opposite side; also, the temporal lobe of the other side and finally the pons.

In addition to the enumerated etiological factors, an abscess in the frontal or in the temporal lobe may be formed on the same or on the opposite side, due to disease of the cerebral wall of the sphenoid cavity and perforation of same, involving the cerebral membranes; furthermore, involvement of the pedunculus

cerebri, the pons, and of the optic nerve of the same or of the opposite side, may ensue. The nerves in the sinus cavernosus and of the orbit may also be affected; finally the arteries and veins may be involved; these will be discussed later. Involvement of the contents of the orbit and their sequellae may be produced by extension of the disease and perforation through the orbital wall of the sphenoid cavity.

Extension of a pathological process by continuity of tissue or direct contact-infection may be greatly favored by the presence of congenital or acquired bone-dehiscence and of a semicanalis ethmoidalis. We found and described a semicanalis ethmoidalis. This semicanal courses in varying lengths from the forumen ethmoidale anterius along the wall of the frontal sinus or the anterior orbital cells to the anterior cranial fossa. In this semicanal the ethmoidal veins are distributed to the respective cavities, liberally protected by the mucosa of the accessory cavities. We will later discuss the relation of the ethmoidal veins to the dural venous network, and the origin of a thrombo-phlebitis. At this point we would merely mention that the accessory sinus mucosa, at these given termini of the semicanal is in contact with the sinus mucosa, the orbital periost and the dura mater; the latter at the point where the semicanalis ethmoidalis terminated in a long fissure in the anterior cranial fossa. In consequence of this relationship, the extension of the inflammatory process may be continued directly to the duramater and the periorbita, in addition to the thrombo-phlebitis of the ethmoid veins. The orbital extension of the inflammation may produce eye complications. In several cases we observed the semicanalis ethmoidalis in the frontal sinus; in three cases the length varied from 5 to 8 mm.; in one case where a confluence of ethmoid cells with the frontal sinus was observed, the 16 mm. long semicanalis ethmoidalis was continued downwards along the lateral wall of the frontal sinus. The length of the semicanalis ethmoidalis varied from 7 to 10 mm. in the anterior fronto-orbital cells, and from 4 to 10 mm. in the posterior fronto-orbital cells.

In congenital or acquired bone-dehiscence the mucosa of the accessory sinuses may come in direct contact with the periorbita, the duramater and the sheath of the optic nerve, and thereby the inflammatory process may readily spread to these specified parts. Then again, the dehiscence of the vessel sulci may lead to disease of their respective vessels.

Congenital physiologic dehiscence of the posterio-cerebral frontal sinus wall were observed by Mouret, Lindt, Castex, Cisneros and

Jaques; Zuckerkandl observed a case of dehiscence, due to senile atrophy of the cerebral frontal sinus wall, of the horizontal plate of the frontal bone, through which the frontal sinus communicated with the anterior cranial cavity. Merlin saw a dehiscence of the superior orbital wall through which the frontal sinus communicated with the orbital cavity. Zukerkandl, Merlin and we observed a dehiscence of the cribriform plate of the ethmoid bone, which opened into the orbital wall of the frontal sinus. Congenital dehiscence of the cribriform plate of the ethmoid was found in three cases by Merlin, in fourteen cases by Zukerkandl, and in eighteen cases by us. We noted that part of the superior wall of the frontal sinus, situated between the lamellae of the superior orbital wall, was much thinner and that part of same contained small round and linear dehiscences. Zukerkandl and Merlin observed dehiscences of the maxillary antrum on the inferior orbital wall. Dehiscence of the canalis opticus was observed by Gillmaertz twice in 200 cases; Holmes twice in 50 cases and by us once in 300 cases. Dehiscence of physiologic origin of the walls of the sphenoid cavity was observed by Zukerkandl as a small cleft in the lateral wall of the sphenoid cavity communicating with the middle cranial fossa. Spee observed in one case a defect in the sulcus caroticus. In a number of skulls we have observed foramina, frequently symmetrical on both sides, immediately below the lateral root of the small wing of the sphenoid. In some of these cases, vessel grooves communicate with the foramina, in which larger or smaller linear dehiscences are found.

Etiologically it is also of importance to consider the strength and thickness of the bony cerebral walls of the accessory cavities; also the density of these walls, and its spongy, vascular diploe. These properties may favor, hinder or check the extension of a disease process, circulation disturbance, bone destruction or fracture. We observed that the thickness of the cerebral wall of the frontal sinus, squamous portion, varied from 1 to 7 mm.; orbital part, from 1 to 4 mm. The thickness of the cerebral walls of the ethmoid cells varied from 1 to 8 mm. The thickness of the superior cerebral wall of the sphenoid cavity varied from 1 to 14 mm.; of the posterior cerebral sphenoid cavity wall varied from 1 to 20 mm. The cerebral walls of the accessory cavity may also measure less than 1 mm., and be extremely thin and of the density of tissue paper. The orbital walls of the accessory cavities are especially thin. Thinnest of all is the cribriform plate of the ethmoid; the orbital walls of the frontal and sphenoidal cavities are somewhat

thicker. The bony partition between the posterior ethmoid cells and the canalis opticus and sulcus opticus is of tissue paper consistency; in several cases it measured from 1 to 2 mm. thick. Between the sphenoid cavity and the canalis opticus and sulcus opticus we have frequently found a strong partition wall, varying in thickness from 1 to 12 mm. It is evident that a poorly vascular, compact cerebral or orbital bony wall, with poorly developed diploe, may offer considerable resistance to direct infection, the extension of a pathological process, the destruction, perforation or fracture of these bony walls, the degree of resistance being dependent on the density and thickness of these walls. The denser and thicker the cerebral and orbital accessory cavity walls, the greater their safeguard and protection against intracranial and orbital complications. Conversely an unusual thinness of the cerebral and orbital accessory cavity walls is to be regarded as a very favorable factor in causing direct infection; likewise the spongy, vascular, thick diploeic layer of the cerebral sinus walls easily permit of affections of the vessels and complications of the meninges and of the brain. The extremely thin bony walls adjacent to the optic nerve favor, to the utmost degree, disturbances in circulation, pressure conditions and inflammatory processes being transmitted to the optic nerve and its sheath, resulting in disturbances of vision and sometimes permanent blindness.

In the etiology of intracranial complications, the vascular system plays an important role in the production of direct and indirect infections. Before discussing this question, let us refer to indirect complications, produced by secondary disease of the accessory sinuses adjacent to the cranial fossa, and of the contents of the orbit.

Just as extension of an inflammatory process along the line of tissue continuity, resulting from direct contact infection, may produce oculo-orbital, intracranial and cerebral complications, so too may the extension of a disease process of a single accessory sinus extend through the associated partition walls to the neighboring accessory cavities and produce there secondary disease. In this way indirect infection of the cranial contents and of the orbital contents may follow secondary disease of the acessory sinuses adjacent to the cranial fossae. The maxillary antrum may have partition walls in common with the sphenoid cavity and ethmoid cells. The common partition wall between the maxillary antrum and the sphenoid cavity we have described and reported in seven cases; the thickness of these walls varied from 4 to 10 mm. The

common wall between maxillary antrum and ethmoid cells we have observed in several cases, and measured 6 to 10 mm. A communication between the maxillary antrum and the posterior ethmoid cells has been described by Zukerkandl; we have described a communication between the maxillary antrum and anterior ethmoid cells. Thus, the pathological process may extend through the common partition wall from the maxillary antrum to the sphenoid cavity; also, from the maxillary antrum to the ethmoid cells, and from these secondary disease processes indirect infection of the cranial cavity may take place. The superior wall of the maxillary antrum, serving as the floor of the orbit, offers a wide field for the transmission of disease processes from the antrum to the orbit. The frontal sinus has partition walls in common with the ethmoid cells and sphenoid cavity; communication may be established between both frontal sinuses by means of a foramen inter-frontali. The frontal sinus may also communicate with the fronto-orbital cells. The first form of communication we observed in one case; the latter in five cases. Secondary disease of the aforementioned cavities may take place through an existing communication as well as by direct extension through a common partition wall. In addition to their relation to the frontal sinus and maxillary antrum, the ethmoid cells have a common partition wall with the sphenoid cavity; usually there is a partition wall between the most posterior ethmoid cell and the sphenoid cavity; we have also observed a common partition wall between the bulla ethmoidalis and the sphenoid cavity. The sphenoid cavity may have partition walls in common with the maxillary antrum, frontal sinus and ethmoid cells. We have also observed a common partition wall between the right sphenoid cavity and the posterior ethmoid cells of both sides; also a common partition wall between the left posterior ethmoid cell and both sphenoid cavities. These common partition walls are usually very thin and are easily diseased.

Both physiologic communications and perforations may influence the neighboring accessory cavities, and by means of vascular communication or by contact infection, may involve the meninges, the brain or the contents of the orbital cavity.

We must associate those venous vessels and venous plexuses which are responsible for the direct and indirect infections of the contents of the cranial cavity and the orbit and also those which may induce disturbances of circulation, emboli, thrombo-phlebitis and thrombi. The fact that the veins of the accessory cavities intercommunicate and also communicate through the bones with the

meninges, has recently been corroborated by Killian. The direct communication with the meningeal venous plexus, as indicated by Zukerkandl,—the connection of the veins of the diploe with the venous network of the dura, as observed by Zukerkandl, Kuhnt and Killian,—the liberal venous distribution in the ethmoid cells, as described by Zukerkandl, and the free distribution of the ethmoidal veins in the semicanalis ethmoidalis, reported by us,—all determine the morphological basis for the development of direct meningeal and cerebral complications.

The more remote venous trunks and networks which are associated with these venous channels may be indirectly responsible for these complications. The veins of the nasal mucosa are associated with the facial veins; the anterior ethmoidal veins are in touch with the dura and piamater, and the venous trunks of the nasal mucosa connect with the palatal and pharynx veins and with the venous plaxus of the pharyngo-palatina. The venous network of the lachrymal canal and of the tear sac is connected with the anterior facial vein, the vena ophthalmica and the vena infraorbitalis. The vena lacrimo-facialis, described by Zukerkandl, communicates with a large venous branch from the anterior ethmoid cells, and finds its way through a foramen in the lachrymal bone. The vena ophthalmica facialis perforates the wall of the maxillary antrum and joins a branch of the venous plexus of the antrum. Kuhnt, Zukerkandl and Gurwitsch have shown that the venae perforantes are connected with the venous network of the dura. The veins of the frontal sinus are distributed along the floor of the frontal sinus and have their outlet in the vena frontalis or in the vena supraorbitalis. The veins of the diploe are in communication with the vena frontalis, with the dural continuation of the foramen coecum and with the sinus longitudinalis superior. The veins of the posterior wall of the frontal sinus are connected with the veins of the dura and with the conus of the foramen coecum. The vena ethmoidalis anterior and posterior empty into the vena opthalmica superior. The vena ethmoidalis anterior may anastamose with the vena angularis and the vena supraorbitalis. We have found the vena ethmoidalis anterior coursing in varying lengths along the semicanalis ethmoidalis in the frontal sinus, and freely in the anterior and in the posterior fronto-orbital cells. The vena ophthalmica superior anastomoses with the venae ethmoidales, the vena centralis retinae and the vena ophthalmica inferior. The veins of the eye connect with the facial veins, the veins of the nasal cavity, the plexus pterygoideus and the sinus cavernosus.

The vena centralis retinae may connect directly with the sinus cavernosus. The veins of the sphenoid cavity may anastamose with the plexus ophthalmicus and the sinus cavernous. Krause has observed the vena ophthalmica inferior or a branch thereof emerging from the fossa pterygo-palatina; also that the central vein of the optic nerve courses through the fissura orbitalis inferior. There is a direct anastamosis between the veins of the several accessory cavity walls, between these and the orbital and dural venous plexus, between the diploe veins of the frontal, the veins of the dura and the sinus longitudinalis, between the ethmoid veins and the piamater, between the veins of the sphenoid cavity and the sinus cavernosus and the plexus ophthalmicus. Indirectly the aforementioned venous anastamoses is facilitated through the veins of the nasal mucosa, the face and the eye.

As previously observed, bacteria have been definitely determined in the vascular channels and around the vessels, thus giving rise to a possible direct infection of the contents of the orbit, the meninges, the brain and the cranial sinuses; furthermore, when disease occurs of the diploe veins, the veins of the accessory sinuses in their anastamoses with the dural and meningeal venous plexus, the exposed venous branches in the ethmoid cells, the exposed ethmoidal veins in the semicanalis ethmoidalis, the sinuses of the skull in their association with the veins of the accessory cavities, a direct infection of the cranial contents, a direct continuation of a thrombo-phlebitis, a thrombus may take place, followed by meningitis, sinus thrombosis and brain abscess; also to emboli of the central vein of the optic nerve, to thrombophlebitis, and thrombi of the eye veins. Disease of the more remote veins and plexuses may indirectly produce oculo-orbital, intracranial and cerebral complications by transmission of a disease process through direct venous anastamosis. Thrombophlebitis of the veins of the maxillary antrum, of the face and of the eye, their thrombi, and the disintegration of the thrombi, may, because of the numerous venous anastamoses with the dural and meningeal venous network, indirectly produce intracranial complications.

In those cases in which the canalis opticus and the sulcus opticus form part of the posterior accessory cavities, where the vessels anastamose with those of the cavity walls, the disturbances in circulation may bring about venous stenosis about the optic nerve, extension of inflammation to the optic nerve and along its sheath, hemorrhage into the sheath of the optic nerve, thrombosis and embolus of the central vessel of the optic nerve, disturbances of

vision and permanent blindness. Inflammation of the contents of the orbit, orbital phlegmon, orbital abscess, dependent on suppurations of the accessory sinuses, may indirectly produce intracranial and cerebral complications; periostitis, ostitis, caries and necrosis of the roof of the orbit, perforation of the bony partition wall between the orbit and anterior cranial fossa, determines the extension of the disease process to the meninges and the brain. The progress of the suppurative periositis along the sheath of the optic nerve and thrombophlebitis of the veins of the eye, may cause infection of the meninges and of the vessels of the cranial cavity.

Besides the above factors, the lymph channels must also be considered in the transmission of infectious material from the nasal cavity and accessory sinuses to the contents of the cranium and orbit.

Schwalbe, Key, Retzius and others report that the subdural and arachnoidal spaces are connected with the perineural sheath of the olfactory nerve, and these are connected with a network of lymph vessels. A definite relation between the perimeningeal spaces and the lymph channels of the olfactory mucosa has been determined in both man and animals; it is also a fact that the perivascular lymph channels of the vessels of the accessory sinuses are not only closely connected, but are also in touch with the venous anastamoses between the accessory cavities and the dura and pia mater.

The micro-organisms found in the nasal cavity must be considered as factors of infection because of their increased virulence.

Howard and Ingersoll designated the diplococcus lanceolatus, streptococcus pyogenes, the bacillus diphtherii and the bacillus influenzae as causes of infection. Holmes emphasizes the presence of the streptococcus and of the pneumococcus in the nasal cavities and in the accessory cavities. Luc found the pneumococcus in a case of acute frontal sinus suppuration. Usually various forms of micro-organisms are found in suppurations of the accessory sinuses. The examinations of Logan Turner, Lewis, Winckler, et al., showed principally the presence of streptococcus. Wolff proved that the diphtheria bacillus was present in all of the accessory sinuses in cases of nasal diphtheria. Isolated cases of bacterium coli were found. Weichselbaum, Dmochovsky, E. Fraenkel and Darling regard the pneumococcus as the most frequent cause of accessory sinus suppuration; with these streptococci and staphylococci are frequently found. The bacillus of influenza is less often found even in cases of influenza empyema. Ostmann found

the diplococcus in the continuity of the tissues; Hajek found the streptococcus in and around the vessels; Killian observed the streptococcus. Gerber, in 65 reported cases, found a marked preponderance of streptococcus; its presence in pure culture was in the ratio of 16 to 7 to the staphylococcus; compared to this all other bacteria are obscured. In complicated cases, Gerber emphasizes the occurrence of staphylococcus aureus. In frontal sinus osteomyelitis, streptococci were found by Luc, Lermoyez, Claone, Laurens and Knapp; staphylococci by Luc, Capart, Botey and Gerber; pneumococci by Luc, Grunert and Gerber. Muller, Gerber, Canon, Guisez, Lannelongue and Achard and Lexer mention the staphylococcus as the cause of osteomyelitis. These etiological factors demonstrate that, when increased virulence is under consideration, the streptococcus pyogenes and the staphylococcus pyogenes are the causes thereof.

To complete our subject, we include herewith, as far as possible, the statistics concerning intracranial and cerebral complications. These statistics are necessarily incomplete. Dreyfuss has published two reports of intracranial and cerebral complications, one to suppurations of the accessory sinuses. St. Clair Thomson contributed to the statistics of suppurations of the sphenoid cavity. Gerber presented an exhaustive report of complications resulting from suppurations of the frontal sinuses. Dreyfuss endeavored to enlarge his first report; his second report was developed by Hajek. The difficulty of compiling complete statistics is best shown by a partial comparison of the chronological additions, which can scarcely be looked upon as final at the present time; for example, the first report of Dreyfuss mentions 13 cases of complications of the sphenoid cavity; St. Clair Thomson added 42 cases; Dreyfuss, in his second report, added 19 cases. As a further example, Dreyfuss mentions 36 cases of brain abscess due to frontal sinus suppuration; Gerber added 30 cases, and we, in turn, have several cases to add. Dreyfuss, in his second statistics, reports 3 cases of successfully operated brain abscess; we had previously in our monograph reported 7 cases of cured brain abscess. This short citation suffices to prove that statistics, notwithstanding most careful work, can not be regarded as complete. The difficulty is due to inaccurate publication, incomplete observation, the absence of post-mortem findings,—facts which are of much importance in the compilation of accurate statistics. Numerous short reports in German literature have been overlooked, as they were not published in extenso.

We mention the few statistical reports of post-mortem findings: In 10,394 post-mortems, Werheim found 127 cases of intracranial suppurations; 53 cases were of otogenic character; in 60 cases there were no details of the source of suppuration; of the 14 remaining cases, 6 were definitely proven intracranial suppuration.

In 6,000 post-mortems, Treitel found 2 cases; in 9,000 Pitt found 2; in 13,400 we found 4 rhinogenic brain abscesses, and besides these four rhinogenic brain abscesses there were 45 otogenic brain complications.

These case records we will utilize and elaborate. We select from these statistics the maxillary antrum and ethmoid cell complications of Dreyfuss, the frontal sinus complications of Gerber, and the sphenoid cavity complications of St. Clair Thomson and Dreyfuss.

Of the maxillary antrum suppurations, Dreyfuss mentions six cases, of which four were brain abscesses. Three of these were in the frontal lobe, one in the temporal lobe, one of meningitis, and one of thrombo-phlebitis of the vena ophthalmica and of the plexus pterygoideus.

Gerber's statistics of frontal sinus complications show: In 473 cases there were 140 brain complications. In five cases of meningitis without brain abscess, there were 14 cases of pachymengitis with extra dural abscess; in 65 cases of brain abscess it was found 25 times; independent extradural abscess occurred in 28 cases; among these the cerebral wall of the frontal sinus was involved in 18 cases. In the 51 cases of meningitis, the anterior wall was involved in nine cases, the floor in two cases, the cerebral wall of the frontal sinus in 20 cases, the septum three times, and in 12 cases these details are missing. Of meningitis serosa, Gerber mentions three cases besides one of Dreyfuss; we add one case which we have observed. Of these five cases, four recovered.

Leptomeningitis purulenta occurred in 51 cases without brain abscess; of these, six with thrombosis; brain abscess with meningitis in 27 cases; of these, two with thrombosis. Thrombophlebitis of the vessels is mentioned in 15 cases. Of these, the sinus longitudinalis superior was involved 12 times. The sinus cavernosus six times, the sinus transversus three times, the sinus sigmoideus once, the sinus petrosus twice, the torcular Herophyli twice, vena ophthalmica five times and the vena frontalis once. To these statistics we add one case of Killian in which frontal sinus suppuration and thrombo-phlebitis of the sinus longitudinalis occurred.

Gerber cites 66 cases of brain abscess following suppurations of the frontal sinuses and eight cases following poly—and pansin-

usitis, in which the influence of the frontal sinus was indefinite. In 44 cases there was involvement of the posterior cerebral frontal sinus wall, especially necrosis and perforation; in 11 cases the floor was also involved; in five cases the anterior and posterior walls were affected; in two cases only the anterior wall; in five cases only the floor; in one case the anterior wall, floor and septum were involved.

There are 50 reports of post-mortem findings. The abscess was most frequently located in the frontal lobes; in three cases there were multiple brain abscesses, once in the temporal lobe, once in the cerebellum and once in the pedunculus cerebri. In Gerber's report, the cases of Blessig and Tilbing, Botey, Finlay and Lack are missing. To Gerber's report we wish to add two cases of Killian and one case of Reiking.

Of the successfully operated and cured brain abscesses, Dreyfuss, in his second report, mentions the three cases of Denker, Rafin and Herzfeld; Gerber mentions seven cases of Denker, Donalies, Gruenwald, Hagen, Hammesfahr, Herzfeld and Rafin; we mentioned in our monograph the seven cases of Denker, Donalies, Hammesfahr, Herzfeld, Killian, Gruenwald and Mueller. With the case of Hagen, mentioned by Gerber, we have, to the present time, eight cured cases of brain abscess.

The report of Dreyfuss on ethmoid sinus complications contains 10 cases of brain abscess in the frontal lobe, 12 cases of meningitis, one case of thrombo-phlebitis of the sinus cavernosus and one case of meningitis serosa. To this we add Huber's case of brain abscess in the frontal lobe.

The report of sphenoid cavity complications of St. Clair Thomson and Dreyfuss records 25 cases of meningitis, 5 cases of thrombo-phlebitis of the sinus cavernosus, one case of thrombo-phlebitis of the sinus longitudinalis superior with meningitis, 17 cases of thrombo-phlebitis with meningitis, 2 cases of extradural abscess with meningitis, 2 cases of meningitis serosa, one case of brain abscess, 3 cases of encephalitis, one case of intracranial hemorrhage. Gerber collected, as previously mentioned, 19 cases of poly—and pansinusitis, among which were 8 cases of brain abscess and 11 cases of meningitis. It may be briefly mentioned that Logan Turner, Dreyfuss and Gerber have referred to the post-operative meningitis, of which 40 cases have been reported. It must be assumed that there are a large number of unreported cases.

Kuhnt's statistics indicate that 93.7 per cent. of the diseases of the lachrymal canal are of rhinogenic origin, traceable principally

to the maxillary antrum, frontal sinus and ethmoid cells. Secondary disease of the eyelids, the conjunctiva and the cornea and nerve reflexes were also caused by affections of the sinuses. Intra-ocular changes, disease of the uveal tract, iritis, cyklitis, chorioditis, detachment of the retina, opacity of the lens, various cataract formations and glaucoma were observed; also emboli and thrombi of the central vessels of the optic nerve.

Birsch-Hirschfeld reports that of 409 cases of inflammations of the orbit, following sinus affections, blindness resulted in 66 cases, or 16 per cent. The most frequently resulting blindness is in cases of empyema of the maxillary antrum, 27 per cent. In 18 cases blindness was due to corneal ulcer, 8 cases to panophthalmia and 2 cases to glaucoma. Death rate is greatest in empyema of the sphenoid cavity, 28 per cent. In frontal sinus empyema it is 16.3 per cent. Post-mortem showed presence of meningitis in 34 cases, brain abscess in 15 cases and sinus thrombosis in 6 cases.

In the etiology of nasal neuritis retrobulbaris, Birsch-Hirschfeld has recently emphasized the vulnerability of the papillomacular bundle of the optic nerve. This increased vulnerability is regarded as the focal point in the changes in the optic nerve in toxic amblyopia. Neuritis axialis, isolated degenerations of the papillomacular bundle, has been proven experimentally and patho-histologically. Mechanical, inflammatory and toxic influences have been considered. Birsch-Hirschfeld regards the central scotoma appearing as the result of injury of the papillomacular bundle as a very important early symptom of tumors and suppurations of the posterior accessory sinuses. There are nine known cases where central scotoma was observed; several are associated with tumors. Birsch-Hirschfeld reports one case of carcinoma in which compression of the vena centralis retinae took place, causing at its posterior point of entrance isolated degeneration of the papillomacular bundle, loosening of the glia fibers and increase and swelling of the glia cells. As the periphery of the optic nerve remained intact, Birsch-Hirschfeld was unable to explain these axial changes as the result of pressure alone, and considers the venous stasis as a toxic influence, a chemical influence of toxic oedema. In my case a sarcoma in the canalis opticus surrounded the optic nerve; the fundus oculi was normal; no examination was made for scotoma. Evidence of pressure in the veins was determined by microscopic examination, but no change was noted in the optic nerve. Examination of a normal optic nerve from the same region gave a very similar picture; the structure of the optic nerve and the number of glia cells

were the same. The significance of Birsch-Hirschfeld's case depends only on a supposition, and is as yet an unsolved question. We mention this observation because of the analogy of Birsch-Hirschfeld's case of a tumor affecting the optic nerve, to ethmoid and sphenoid cavity inflammations with the presence of a central scotoma.

Without doubt, the acceptance of the question of increased vulnerability of the papillomacular bundle easily explains disturbances of vision of nasal origin, and must be regarded as an important clinical symptom of diseases of the posterior accessory sinuses. Fuchs has observed that acute neuritis retrobulbaris with central scotoma frequently occurs as the result of coryza and influenza; accessory sinus disease does not seem to be a necessary factor; often the rhinological data are negative, nor need toxaemia be present. According to Fuchs, the papillomacular bundle is not only toxically affected, but hyperaemia or swelling of the sheath of the optic nerve in retrobulbar neuritis, in diseases of the accessory sinuses or pressure from tumors may also be present; that the bundle is not especially injured is probably due to the fact that acute retrobulbar neuritis so often results in entire restoration of vision. In his most recent contribution, Fuchs also remarks that the papillomacular bundle is liable to disease because of its great vulnerability.

The increased vulnerability of the papillomacular bundle explains its susceptibility and the development of a central scotoma and the eventual degeneration of the optic nerve in diseases of the nasal cavity and of the accessory sinuses. These injuries may be caused by disturbances of the circulation, venous stasis, extension of inflammation, pressure, and finally suppuration and toxaemia due to malignant tumors.

In toxaemia, a progressive affection of the optic nerve must also be taken for granted, but this question cannot be discussed here. The section of the optic nerve of the canalis opticus supplied by the recurrent branches of the arteria and vena centralis retinae may injure the sensitive bundle through circulatory disturbances even when the accessory sinuses are not involved. In the section of the optic nerve in the canalis opticus, hyperaemia, oedema, and inflammation of the sheath of the optic nerve is especially prone to produce dangerous pressure or venous stasis in the optic nerve in the rigid-walled area of the canalis opticus. Venous stasis in the nasal cavity and in the accessory sinuses may indirectly affect the vena centralis retinae and the vena centralis posterior. The fact so frequently observed by us that the canalis opticus forms the

supplementary part of the posterior accessory sinuses, is often freely exposed, contains in the semicanalis ethmoidalis the exposed venae ethmoidalis and an exposed venous branch of the ethmoid cells (Zukerkandl), may easily explain circulation disturbances and the extension of inflammation in the part of the optic nerve and its sheath that lies in the canalis opticus and also injury to the sensitive papillomacular bundle. In all cases where the canalis opticus forms a part of a posterior accessory cavity or is prominently present in such a cavity, the blood and lymph vessels are in close relation to the sinus walls, and when such a cavity becomes diseased, circulation disturbances are likely to follow.

Our investigations have proven that in the domain of the optic nerve the canalis opticus is usually protected from the ethmoid cells and especially from the sphenoid cavity by a bony wall frequently of 12 mm. thickness; then too, the venous drain of the optic nerve may be very free in those cases where the vena centralis retinae empties freely into the sinus cavernosus or into the fissura orbitalis inferior. There may therefore be structural conditions unfavorable to venous stasis in the optic nerve, extension of inflammation in the region of the canalis opticus, of the optic nerve and its sheath, which may insure the intactness of vision. Thus we explain the absence of disturbances of vision in the presence of suppuration and destruction of the bony walls of the sphenoid cavity. Baratoux describes a case in which the greater part of the body of the sphenoid bone was exfoliated without producing serious disturbances; Foucher describes a case of necrosis of the sphenoid cavity and bone sequestration; the patient died; no disturbances of vision could be observed.

On the strength of our observations we are opposed to the stereotyped view which endeavors to associate diseases of the sphenoid cavity with canalicular affections of the optic nerve; in many cases the sphenoid cavity has nothing in common with the optic nerve, as the closer association exists only with the posterior ethmoid cells. Where both the ethmoid and sphenoid cells are simultaneously affected, both cavities must be considered, and under no circumstances should the sphenoid cavity alone be regarded as the causal factor. Our morphological findings indicate that an extension of inflammation is possible by way of the posterior ethmoid cells as by the sphenoid cavity, and also by disturbances in circulation.

Mendel and Lapersonne assert that a unilateral neuritis is characteristically of nasal origin. Birsch-Hirschfeld believes, in unilateral disease of the posterior accessory sinuses, that a differential diagnosis points to disease of the optic nerve and against a toxic

and infectious neuritis optica, even though bilateral disease may occur. Mendel and Lapersonne regard bilateral neuritis to be of intracranial origin. Sattler thinks that a unilateral neuritis optica, relatively atrophia nervi optica, is in no way characteristic of sphenoidal and ethmoidal disease; such a unilateral neuritis may result from inflammation, hemorrhages, tumors at the cerebral terminus of the canalis opticus or toxic influences. In the majority of the cases thus far observed, disturbances of vision and blindness occurred on the same side as the unilateral sinusitis. The number of cases of bilateral suppurative sinusitis and disturbances of vision is much smaller; the cases of bilateral disturbances of vision with unilateral sinusitis are also less frequent.

Our reported morphological findings explain circulation disturbances and lesions of both optic nerves and chiasma in unilateral affections of the sphenoid or posterior ethmoid cells. Individual cases and our anatomical investigations point conclusively to the fact that both optic nerves may be influenced by unilateral sinus disease. There are reports of 100 clinical cases. Many are of little value, because details are wanting.

We know of cases in our literature that rest only upon assumption, or where the further development of the cases would require additional data. Numerous cases have been reported with improvement of vision following treatment of the posterior accessory sinuses, but the termination of these cases is not reported. It would be essential to know whether these cases were cured by treatment and operation or whether these measures were futile.

Many cases are cited in literature of serious complications of thrombo-phlebitis of the veins of the eye and the vessels of the cranial cavity, valueless because neither clinical nor post-mortem investigations were made. We have access to twenty cases of thrombo-phlebitis of the vena ophthalmica, the sinus cavernosus, sinus circularis, sinus petrosus and sinus longitudinalis, following empyema of the sphenoid cavity.

In the clinical experience of all rhinologists there are many cases of less or greater disturbances in the posterior accessory cavities where no changes in vision occurred.

We have previously called attention to those conspicuous cases where tumors in the nasal cavity, empyema and extensive necrosis have not affected vision. Spontaneous recovery may follow empyema; a case of neuritis reported by Copez and Lor, recovered, notwithstanding that the sphenoid cavity was not opened. Neuritis may also disappear with the spontaneous recovery of acute empyema, just as central scotoma may disappear when rhinitis sub-

sides. There are cases reported where endonasal, conservative treatment relieved the disturbances of vision.

There are a goodly number of operated cases, with recovery, of unilateral, bilateral and contralateral disturbances of vision as well as oculo-motor paralysis.

There are also cases where either the unfavorable neuritis could not be checked or a total atrophy of the optic nerve remained unchanged, due to long-standing empyema or to other accidental causes.

Of contralateral disturbances of vision we have no post-mortem records; the conditions which we have described constitute the anatomic basis, explaining lesions of the contralateral optic nerve; also of the chiasm of both optic nerves, unilateral disease of the posterior ethmoid cells and of the sphenoid cavity. Illustrative of these conditions are the cases of Wohlmuth, Freudenthal, Lors and Levy, Kuttner and Lehmann, Halstead, Glegg and Hay, Pollatsek, Mayer and my own case.

In the first four cases there was a fracture of the contralateral canalis opticus; the last five cases depend on empyema of the posterior accessory sinuses. Indirect fracture of the canalis opticus of the same side has been more frequently observed. Hoelder has found fracture of the walls of the canalis opticus in post-mortem examination of 53 cases.

Finally we must mention as an etiologic factor, artificially produced disturbance of vision during operation. This occurred in a case of Hirschfeld's in which a superior semi-blindness of the papillae followed operation for empyema of the right maxillary antrum, ethmoid and sphenoid cells.

This traumatic lesion of the optic nerve occurred while curetting the ethmoid and sphenoid cells by way of the maxillary antrum. This accidental trauma was due to the extension of the canalis opticus into the posterior ethmoid cells and the sphenoid cavity, as we have previously described. In conclusion we would observe that many cases have been followed by rhinogenic blindness.

Undoubtedly the extension of the posterior ethmoid cells and of the sphenoid cavity in the region of these nerve trunks is responsible for the lesions and participation of the oculomotor nerves and of the trigeminus in diseases of the accessory sinuses. The posterior ethmoid cells and the sphenoid cavity may, at times, be extended to the lesser as well as to the greater wings of the sphenoid, and may, in this manner, come into closer relation to N. trochlearis, N. oculomotorius, N. abducens and the trigeminus. Paralysis of the oculo-motor nerves occur in caries, necrosis of the superior and

lateral walls of the sphenoid cavity, in thrombophlebitis of the sinus cavernosus, in intra-cranial suppurations and in tumors. In cases where an unusually thin bony partition separates the nerves from the posterior ethmoid cells and the sphenoid cavity symptoms of paralysis and sensory disturbances may be caused by disease of the posterior cavities. The lateral wall of the sella turcica may, according to my observation, be formed entirely of the sphenoid cavity or partly of the posterior ethmoid cells; furthermore, both cavities may bound the fissura orbitalis superior and the foramen rotundum and ovale. Through the fissura pass the N. oculomotorius, N. trochlearis, a branch of the trigeminus and the abducens; through the foramen rotundum the second branch of the trigeminus leaves the cranial cavity; through the foramen ovale passes the third branch. In empyema of the ethmoid cells and sphenoid cavity, Thomson observed total paralysis of the N. oculomotorius. In sphenoid cavity empyema Hoffman, Lapersonne and Stanculeanu found total paralysis of N. oculo-motorius; Baumgarten paralysis of N. oculo-motorius; Panas, abducens paralysis and trigeminus anasthesia; Mahn, abducens paralysis; Fisch, paralysis of the adducens and abducens. Panas found anaesthesia of the second branch of the trigeminus; Rouge, intra-orbital neuralgia; Schaefer, Moreau, Hajek and Schroeder, supra orbital neuralgia.

In empyema of the ethmoid cells, asthenopia was observed by Burger, Caldwell, Hajek and Gruenwald; accommodative asthenopia was observed by Moreau and Schroeder, following sphenoid cavity empyema; Lapersonne and Fisch have called attention to increased pressure of the eyeball.

The infrequency of paralysis in accessory sinus disease is due to two causes: First, the rare occurrence of a close association between the posterior ethmoid cells and the nerve roots; again, a consideration of the thick bony protecting wall where the sphenoid cavity frequently comes into closer relations with the nerve roots. Second, the claim of Fuchs that the frequent escape from injury of the fibers of the inner muscles in peripheral processes is due to their lesser vulnerability as contrasted with the greater vulnerability of the papillomacular bundle of the optic nerve.

Kuhnt, German, Ziem, Varese, Harlan and Bryan consider the field of vision too narrow as an accompanying symptom; Hinkel, in 20 cases, and Haeffner and Henrici, in 37 cases, found no narrowing of the peripheral field of vision.

Of the 100 reports at our disposal of nuritis retrobulbaris and atrophia nervi optici dependent on diseases of the accessory sinuses,

there are many records which must be discarded because of the lack of careful examination and clinical data.

There can be no doubt of the causal relation between diseases of the posterior accessory cavities and disturbances of vision, yet we must emphasize that both conditions may occur simultaneously, accidentally but independently. Ophthalmologists have cited numerous causes for neuritis and atrophy of the optic nerve. Neuritis retrobulbaris may also recover spontaneously after simple treatment or without therapeutic measures. The case of Coppez and Lor is especially worthy of mention, because the neuritis optica following chronic empyema of the sphenoid cavity was healed without surgical intervention; there are cases, such as those of Lapersonne and Gronbaeck, where the unfavorable progress of the neuritis optica was unchecked by operative measures; we know of cases where simply the clinical diagnosis of neuritis or atrophia optica was made by the ophthalmologist without the etiology having been determined, and where rhinological examination was negative. Finally, there are cases of tumors of the nasal cavity, empyema of the posterior accessory cavities and extensive bone destruction where vision remains undisturbed.

We will endeavor, on another occasion, to prove the remarkable fact that no disturbances of vision occur with extensive pathological processes, while, on the other hand, slight disturbances in the nasal cavity may cause disturbances of vision. We would emphasize the fact that disturbances of vision or blindness arising from other causes may be independent of empyema of the posterior accessory sinuses. The simultaneous but accidental affection of these areas may lead to error, and may be detrimental to a clear understanding and development of this interesting question.

The morphologic data, the macroscopic pathologic-anatomic findings, the pathologic and bacteriologic examinations, form the basis of the subject of oculo-orbital, intracranial and cerebral complications following suppuration of the nasal accessory sinuses. The cited statistics indicate the large number of complications and their unfavorable termination. The direct and indirect infection, contact infection of the cranial and orbital cavity contents and the avenues of infection may thus be explained. Detailed pathohistologic and bacteriologic examinations are accessible in but few cases and a more accurate diagnosis in individual cases is desirable because of their importance as contributions to the upbuilding of this subject; a more careful pathohistologic and bacteriologic investigation of the tissues in their continuity and of the vessel, is required to solve the many open questions in this field.

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THE PRESENT STATUS OF SURGICAL TREATMENT OF CHRONIC SUPPURATIVE DISEASE OF THE NASAL AND OF THE AURAL CELLULAR SPACES—A COMPARISON.*

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Men working in any limited special field of medicine are apt to be so engrossed with the requirements of their chosen branch as to be apathetic about investigating, or even noticing, the changes and advances made in some other and perhaps closely-allied field, and to overlook the fact that improvements and discoveries in allied specialties may be with great advantage made applicable to their own. Because of this, one special branch often lingers far behind another, waiting in the dawn of the morning, as it were, while the knowledge gained in a kindred branch, if propertly recognized and applied, might be utilized to push that branch on to the very height of attainment, and so, though advanced knowledge has been gained by such co-workers, the wisdom of applying it promptly to other similar needs is not made use of. "The knowledge comes, but wisdom in its application lingers."

In studying the pathological processes which occur in the nasal and the aural cellular spaces, one may be at times impressed with the apparent analogy existing between the two conditions and may be led to draw a comparison as to the methods of treatment employed by the rhinologist on the one hand and the otologist on the other. From such a comparison it would seem that while the aurist has now very clearly marked out what constitutes proper surgical treatment of the aural cellular spaces, the rhinologist is still floundering about in the dark amidst great diversity of opinion as to the indications for surgical treatment and as to what constitutes proper surgical treatment in chronic suppurative disease of the nasal cellular spaces.

In an anatomical and pathological comparison of the nasal and the aural cellular spaces we find that the cellular spaces of the ear subject to chronic purulent inflammation consist of the tympanum, labyrinth, mastoid antrum, and the mastoid mass of small pneumatic cellular spaces, this latter being a labyrinth of cellular bony substance comprising almost the entire subcortical structure of the

*Read at the meeting of the American Laryngological, Rhinological and Otological Society, Atlantic City, June 5, 1909.

mastoid portion and often extending into the squamous portion of the temporal bone. The middle ear, or tympanum, with its continuation into the mastoid antrum, may be regarded as in one sense but another nasal accessory sinus, communicating with the nasal cavities proper by the naso-pharyngeal tube. Anatomically and histologically these structures are very similar, consisting of bony pneumatic cavities lined by an invagination of the naso-pharyngeal mucous membrane, and pathologically liable to the same inflammatory and suppurative processes.

The mastoid antrum itself varies in size and position as it continues into the mass of smaller cellular spaces behind and above, while the small pneumatic cells may not be confined to the mastoid portion, but may be found in the posterior root of the zygoma, squamous plate of the temporal, in front of the external auditory meatus, and even extending into the occipital bone. The lining membrane of all these cells consists of non-ciliated pavement epithelium, serving both as lining membrane and periosteum. In the larger space of the antrum this mucous membrane is considerably thicker, forming with its reduplications a means of closure of the mastoid from the tympanic cavity under conditions of sudden or prolonged congestion. The bone tissue separating and surrounding these pneumatic cells is, according to Blake, "very vascular, soft and friable, and when invaded by a septic process little able to withstand or to limit it. (Blake and Reik, p. 22-34.)

The cellular spaces of the nose consist of the large accessory sinuses, frontal, sphenoidal, and maxillary, and of the ethmoidal labyrinth or lateral mass of small pneumatic cellular spaces. The larger cavities, like that of the larger aural cavity—the tympanum, are lined with a continuation of the naso-pharyngeal epithelium, while the smaller spaces, the lateral ethmoidal mass of cells, like the mass of mastoid cells, are lined with non-ciliated pavement epithelium and are likewise "vascular, soft and friable," and therefore non-resistant to the invasion of a septic process, the walls of these cells being as thin as tissue paper. According to Woakes, these ethmoidal cells are coated on either side with periosteum protected merely by a thin, delicate epithelium—while, according to Blake, the lining of the mastoid cells likewise "serves both as lining membrane and as periosteum."

Comparing the larger nasal and aural cellular spaces we have also an analogy in anatomical as well as histological structure; both

the tympanum and the mastoid antrum, like the sphenoid and maxillary, are large irregular cavities, having their natural opening or outlet higher than their floors, thus being most disadvantageously placed for drainage, so that acute inflammation, purulent infection and retention of pus is favored mechanically in all these cavities. The same condition of things would seem, moreover, to exist in the masses of small cellular spaces; the mastoid cells once invaded by purulent infection have, of course, very inadequate drainage and little power of resistance, while the ethmoidal mass of cells, likewise, crowded in as it is into the upper angle of the nasal space, under the middle turbinate, suffers the same disadvantages. While the frontal sinus would seem to be more fortunately situated anatomically with reference to drainage, it is to be remembered that it, too, drains only by a tortuous canal easily closed by inflammatory swelling of the ethmoid cells, middle turbinate body, or its own mucous lining. Even with free drainage purulent inflammation and necrosis of the bony walls of these spaces may occur, although, of course, more apt to follow the retention of pus. According to modern writers, too, these masses of ethmoid cells are considered as probably but the residual remains of the olfactory organs, degenerate bodies therefore, very liable to inflammatory processes, the tissues being functionless and of low vitality.

The treatment of acute purulent disease of the larger nasal cavities is quite similar to that of the larger aural cavities—that is, incision or puncture, and the establishment of free drainage—but, when it comes to the treatment of the chronic suppurative conditions, especially those of the lateral ethmoidal mass of cells, which corresponds in so many respects to the mastoid mass of pneumatic cells, the treatment, at least with the majority of rhinologists, is not the same, but may rather be compared to the surgical treatment accorded the latter region fifteen or twenty years ago when the otologist considered that to make a puncture, drill a hole into the mastoid antrum and establish drainage was all sufficient. For a long while the simple Wilde's incision was considered by most otologists as all that was necessary or justifiable. When some bolder otologists began to go further and search out all the diseased mastoid spaces, endeavoring to completely erase the disease focus, they were considered extremely radical, but finally the necessity for such procedure became well understood and appreciated, and now nothing short of complete exenteration of all these aural cellular spaces in the mastoid operation for the cure of chronic suppurative

disease is deemed sufficient, indeed, nothing short of that is considered good surgery. Furthermore, for the cure of chronic suppurative disease of long standing, a complete exenteration of all the adjacent communicating spaces is considered justifiable and necessary—the so-called radical mastoid operation.

Notwithstanding this advance in aural surgery, it is certainly true that most rhinologists still treat chronic suppurative disease of the nasal cellular spaces, especially of the lateral ethmoid mass of cells and frontal sinus, by methods that may be compared to these early and abandoned methods of the otologists; that is, they simply resect the anterior end of the middle turbinate and perhaps curette away a few of the anterior ethmoidal cells, expecting by the better drainage thus supposed to be established to bring about a cure of the suppurative and necrotic disease above, a forlorn hope certainly, when just as in the case of the diseased mastoid mass of cells, nothing short of complete eradication of the necrotic mass should be expected to cure. No doubt, in many cases so treated, the patients are made more comfortable, but obviously, cure cannot be expected merely from better drainage any more than it could be in a similar treatment of the mastoid cells, for unless the exenteration of this mass of diseased cells is complete, the remaining necrotic foci continue the suppuration and even infect the newly exposed portions. It is a well understood principle of surgery that where there is necrotic tissue in a cavity it must be thoroughly removed before a cure can be expected. To simply establish better drainage and trust to nature for the rest is not in accordance with our modern ideas of surgery.

Perhaps in no other particular condition is this difference between aural and nasal surgical methods better shown than in the treatment of polypi. The rhinologist still lassoes the nasal polyp with the wire snare and removes it by traction, leaving the necrotic base from which it grows untouched, or merely curetted a little, a method abandoned long since by most otologists, who believe that aside from being an inefficient method, this is one attended by considerably more danger than the radical operation, many cases of meningitis having been reported as following the removal of aural polypi in this way. We may recall that Dr. Clarence J. Blake and Dr. Hiram Woods, some years ago warned against this procedure in aural surgery as being very dangerous, and now some otologists prefer to do the radical operation when there are existing polypi, which always means necrotic bone. It would seem that the same

principle should apply to nasal polypi. In writing of this subject many years ago, Woakes pointed out that chronic suppurative inflammation in the ethmoid region always passes from a chronic osteitis to caries of the bone, and, though Mr. Waggett in commenting upon this idea says, "The disease finds no parallel elsewhere in the human skeleton, which is not surprising when you remember that the conditions of the ethmoid bone are unique, as nowhere else do you find thin plates of bone coated on either side with periosteum and protected from bacterial influences merely by a thin delicate epithelium," otological writers have pointed out the exactly similar formation of the aural cellular spaces and the fact that they too are subject to what Dr. Blake has called a "rarifying osteitis" and polypoid formation. Though the condition of nasal polypus is usually one that if let alone or not attacked radically runs a long course, yet the rapidity with which it may pass from the initial to the final stage has been noted by Mr. Waggett, and he, though a very conservative surgeon, advises radical procedure for the cure of this condition, the operation consisting in the complete exenteration of the whole lateral ethmoidal mass, just as the otologists now exenterate the entire mastoid cellular mass for the similar condition.

How many of these cases of intranasal suppuration, especially of the ethmoid mass of cells and frontal sinus, are allowed to go on to the development of intra-cranial complication by these methods of timid and incomplete surgery, just as the cases of suppurative involvement of the mastoid mass of cells were formerly allowed to go, it would be impossible to estimate. It is probable that in many of these cases of chronic suppurative ethmoidal and frontal disease, the dura has been in direct contact with the purulent secretions for a long time and that it is only because of its strong power of resistance that a fatality is postponed.

Mr. Balance, in his work on brain surgery, calls attention to the fact that in the cases of otogenetic meningitis, fatal invasion of the dura may be long postponed even when the dura is in direct contact with the suppurative area. The only way of preventing the possibility of extension to the intra-cranial cavity either in the case of aural or nasal suppurative disease of this kind is certainly by complete and thorough removal of the disease focus. That its accomplishment in the case of the nasal cellular spaces is more difficult and perhaps attended by more immediate danger as an operation because of the obscurity of the operative field and its

difficulty of access, should certainly not be allowed to subvert the proper surgical procedure.

Mr. Balance also further draws a very impressive comparison between the advanced methods of otologists and the backward methods of rhinologists when he says, "The importance of effectually dealing with temporal bone suppuration is now fairly well known and the operation for its relief has slowly become appreciated, though retrograde papers on the subject continue to appear, but in this country the radical treatment of frontal and ethmoidal suppurative disease is not thoroughly carried out. Acute cases are sometimes left until the patient has developed meningitis, while in chronic cases the danger of the disease is not even recognized and is apt therefore to be left unremoved. Chronic suppuration in the accessory cavities of the nose is exactly comparable to temporal bone suppuration and, like it, should be treated strictly in accordance with the ordinary surgical principles applicable to the treatment of diseased bone wherever situated, namely, complete ablation."

Thoroughness of operative procedure is the *sine qua non* for success in these cases and it should ever be borne in mind that just as the aural pneumatic cells are not confined to the mastoid, but may extend into the surrounding parts, so the ethmoidal pneumatic cells may not be confined to what is regarded as the ethmoidal region proper, but that they must be searched for in the same painstaking manner employed by the otologist. The aural surgeon considers it necessary to make the most careful search for all the pneumatic spaces before concluding his operation, in order to leave no suppurative foci, and yet the mastoid cells are not nearly as systematically arranged as are the ethmoid cells, and it is perhaps more difficult to reach them all. It is even reported by Dr. Wales that in one temporal bone specimen a mastoid cell communicated with the sphenoid sinus.

It is probable that frontal and ethmoidal suppuration are as often productive of meningitis as is temporal bone suppuration, because of the anatomical structure of the parts and because of the fact that the nasal space is a favorite breeding-place for the meningococci and the pneumococci, and these meningitis producers are so often able to invade the cranial cavity from the nasal space even in the absence of obvious nasal disease. Although the available statistics would probably indicate that suppurative aural disease is responsible for many more cases of intra-cranial complication than

suppurative nasal disease, there is not much reliance to be placed in them for, as Logan Turner has pointed out, post-mortem examinations of the nose have not been made systematically and for a long time as have those of the aural region, though recently more attention has been paid to the nasal cavities as a cause of such infections.

As the object of the radical mastoid operation is to convert the aural cellular spaces into one freely opened cavity and to remove all the morbid tissue, so the object of the radical intra-nasal operation should be to get rid of all morbid tissue and establish free, roomy, nasal space. These have been laid down as cardinal principles in aural surgery, why not in nasal? Whether or not all the ethmoidal cells can be reached as a rule, may be questionable, but that argument was also formerly used against the procedure of thorough mastoid operation. It is certainly possible in most cases to eradicate all the diseased tissue and the attempt should always be made by the rhinologist as well as by the otologist to accomplish this. It may be said that the function of the ear being only that of an organ of special sense, the destruction of which does not affect life itself, it differs from the nasal function, the destruction of which may menace life; but, life is already menaced by the mere existence of a chronic suppurative nasal disease which itself impairs the function of the nose, both as a special sense organ and as a respiratory organ. In fact, chronic purulent nasal sinusitis is a greater menace to life than any degree of interference with nasal respiratory function can be. We have been timid about doing radical surgery in the nasal fossae largely through fear of destroying the respiratory function, but in these cases in which such treatment is indicated, any functional activity that may be possibly attributed physiologically to these intra-nasal structures has either been destroyed completely or so impaired as to render such an objection a negligible quantity. Whether the ethmoidal cells and middle turbinates are physiologically functionless, as is now believed by many, or not, is, of course, still a matter for difference of opinion, but certain it is that when they have become involved in purulent and necrotic disease, they cease to be of value in the respiratory function and can only act as obstructions to proper ventilation and drainage and there can be no good reason, either practical or theoretical, for their retention.

Radical procedure along this line has perhaps been too long retarded and discouraged by an undue respect for precedent. We have been too long content to follow in the beaten paths of our

predecessors in rhinological work. Without being lacking in proper respect for the traditions of rhinology, one may certainly honestly doubt the efficacy of the older methods of treatment and so investigate and work out new developments. Because of the traditions regarding the almost divine function attributed to these intra-nasal tissues, especially to the turbinated bodies, whose removal becomes necessary in radical intra-nasal surgery, traditions which with some rhinologists seems to amount to almost a superstition, scientific investigation has been slow to penetrate the obscurity in which the disease of these intra-nasal cellular spaces has been held. Certainly when one has seen many of these cases operated upon radically, with success he can scarcely any longer endorse the views held by some of the so-called conservative men and recently expressed by one eminent rhinologist in these words, that, "These bodies are not only essential to health, but to life itself."

To quote once more from Mr. Balance, "The intra-meatal aural specialist of a past generation was content to flit helplessly about his chosen canal in the manifest presence of lethal complications. Is it, or is it not true that the intra-nasal specialist of the present day, with some brilliant exceptions, may at times be unduly influenced by the traditions of his otological kinsmen instead of following the teaching of Killian and facing the operation for the complete removal of the disease? Operation for the cure of ethmoidal and frontal suppuration is now regarded in this country much in the same way as was the mastoid operation twenty years ago, as if the lesson that danger attends delay and imperfect operation had to be learned."

We know, too, that although the chances of recovery are, of course, very slight after meningitis has once developed, yet in otological practice many cases are recorded in which there has been recovery after the eradication of the local focus of disease, and that now the otologist feels justified in taking these radical procedures for the cure of chronic suppurative otitis simply as a preventative operation, looking upon a chronically discharging ear as a constant menace to life. Is not a chronically discharging nasal sinusitis just as much a menace to the life of the individual, and is it not just as necessary to endeavor to remove the disease focus in these cases?

However, notwithstanding these indications, as pointed out by Mr. Balance and others, papers continue to appear advocating more

conservative treatment of the nasal cellular spaces, just as they did for so long concerning the treatment of the mastoid cellular spaces, though it would be difficult now to find an aural specialist who would make a plea for less radical treatment of chronic suppurative otitis, and, it is to be hoped that as our knowledge of the cellular nasal spaces and our nasal surgical technique improves, we may soon be beyond the accusation of timid surgery and that we shall advance to the radical work for the cure of these chronic intra-nasal suppurative conditions as our co-workers the otologists have to the cure of similar aural conditions.

With some rhinologists radical operation for disease of the sinuses is considered necessary only when there is evidence of intra-cranial or ocular complications, while the otologists consider such operations indicated not only under these conditions, but whenever there is a long-continued purulent discharge associated with discomfort or pain or there are indications of bone necrosis, or, of course, a combination of these symptoms. Whether the radical treatment of the nasal cellular spaces should be intra-nasal or extra-nasal is, of course, not pertinent to this discussion. Each case must be treated individually, taking into consideration the patient's feeling about the matter as regards cosmetic effects, but as a rule, except in such a sinus involvement as may be complicated by evidence of intra-cranial invasion, frontal bulging or a fistula, intra-nasal radical operation should be adopted first—but it should be radical, and in considering surgical procedure in these cases, we should certainly endeavor to avoid that blind conservatism which so long retarded the advance of thorough otological surgery, that blind conservatism which so often begets in its turn a blind radicalism.

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FACIAL PARALYSIS DUE TO AURAL LESIONS.*

BY D. J. GIBB WISHART, M. D., TORONTO.

The Facial Nerve in its course through the Fallopian canal lies entirely within the Petrous bone and pursues a devious path which leads, first—outward in the direction of the inner wall of the middle ear, as far as the Hiatus Fallopii. Here at the Geniculate Ganglion it turns at an acute angle, and runs for 10 mm. in an oblique direction backwards, inclining at the same time outwards and downwards, along the inner wall of the middle ear, upon the posterior half of which it forms a slight bulge above the foramen ovale, just below the roof, to a point in front of the middle of the aditus between the antrum and the posterior wall of the middle ear, where it is superficial. Bending at an obtuse angle, *on a level with the foramen ovale, and 3 mm. behind* it, the nerve descends in what is practically a vertically straight course to leave the bone at the Stylo-Mastoid Foramen. The nerve, however, does not always adhere to its normal path, for Dench states that the nerve may pass obliquely through the mastoid, becoming quite superficial just before it enters the Stylo-Mastoid Foramen, a statement borne out by Case VI. Loeb pictures a dissection where the nerve at the obtuse angle lay considerably internal to the floor of the aditus. Prentiss and Dean describe a case where the facial nerve entered the posterior surface of the Petrous bone, passed outwards under the superior semi-circular canal, and turned vertically downwards in the same plane, having no relation with the middle ear or antrum.

In its course through the latter two-thirds of the Fallopian canal *two branches emerge* from the main trunk—that to the Stapedius muscle, which emerges opposite to the pyramid, immediately behind the Fenestra Ovalis; and the Chorda Tympani, which arises about 6 mm. above the Stylo-Mastoid Foramen from the descending portion of the nerve at the back of the tympanum.

The character of the bone which covers the nerve in the latter two-thirds of its course must be noted carefully. In infants, the outer wall of the horizontal portion and elbow is a thin diaphanous plate, and even in adults this plate may be extremely thin, but is

*Read before the Canadian Medical Association, 42nd Annual Meeting, August, 1909, Winnipeg, Manitoba.

usually solidly eburnated. The descending portion is contained in a compact lamina, arched praemastoid lamina, but this is thinned in the partition which separates the jugular fossa from the Fallopian canal, to a frail condition, by large pneumatic cells.

The Fallopian canal *communicates* permanently with the middle ear cavity by (1) the aperture of the Stapedius muscle and its nerve; (2) the channel of the Chorda Tympani; and (3) the canal which contains the Tympanic branch of the Stylo-Mastoid branch of the posterior auricular artery; in infancy there is (4) a defect in the bony wall of considerable size just over the foramen ovale. In addition to these there is frequently present in adult life a hiatus in the horizontal part of the canal.

The *distribution* of the facial nerve is perhaps best outlined by a consideration of the indications of a facial paralysis. These are present when the face is in repose, but are exaggerated markedly during facial movement. In the former instance, the affected side is smooth and expressionless, the brow is unwrinkled, and the naso-labial fold obliterated. The palpebral fissure is widened, the eye cannot be closed, and the vision is dimmed by the flooding of the eye with tears. The anterior naris on the affected side is narrowed, and the tip of the nose is deviated to the opposite side. The mouth appears twisted, but the uvula and the palate are unaffected in uncomplicated cases. (Risien Russell).

In the latter the patient cannot wink with the eye, whistle, blow out a candle, or inflate the cheeks. In laughing, crying or speaking, the twisting of the mouth is made more evident. The labial sounds are imperfectly formed, and the speech is therefore indistinct. Mastication is interfered with by the loss of power in the Buccinator muscle, food lodges in the cheek, and the saliva escapes from the corner of the mouth. The tongue may also appear to deviate on protrusion. Smell is impaired from lack of power to dilate the nostril, and from the dryness of the mucous membrane.—(Macewen).

If the paralysis involve the Stapedius muscle, there is hyperacusis, a strengthening of the lower tuning forks. Subjective noises may be present also from the loss of control of the labyrinthine pressure.

Other more remote symptoms sometimes noted are anomalies in certain secretory functions—the saliva may be decreased or greatly increased, tears may be scanty or wholly lacking, and the action of the sweat glands altered.

Disturbances of sensation on the affected side, and even the onset of herpes on the uvula, palate and tongue have been reported.

Severe pains in the head, in the forehead, temple, ear and neck may precede or accompany the onset of paralysis.

Finally trophic changes in the muscles and facial bones may take place, and along with these, the reaction of degeneration in the nerve fibers themselves may be elicited and the longer the degeneration proceeds both Faradic and Galvanic irritability gradually diminish. If paralysis occur in a young child the subject of middle-ear disease, arrest of growth of the affected side is apt to ensue, leading to further disfigurement.

Paralysis, partial or complete, of the facial nerve, *may occur* in the course of any inflammatory process of the middle or internal ear. These processes embrace a rather wide area of affections, of which I will indicate only a few.

When otitic in origin, the paralysis is always of the peripheral type.

A neuritis, or perineuritis, or parenchymatous neuritis, without interstitial changes, is probably present where in the absence of obvious aural lesions recent special exposure to cold produces paralysis. A draught, an attack of rheumatism, etc., may be assigned as the cause. In the latter, Hunter Tod ascribes the resultant paresis to pressure upon the nerve trunk, from enlargement of glands, or some inflammatory thickening around the Stylo-Mastoid foramen; while, in the former, Burnett believes an acute Otitis Media is present. Edward Jackson in Posey and Spiller, "The Eye and Nervous System," speaks of the neuritis as toxic in origin in syphilis, rheumatism, diabetes, gout, leukaemia, diphtheria, etc.

Effusions into the Fallopian canal, due either to abnormal pressure from pent-up secretions finding their way through one of the anatomical openings in the bony wall above referred to, or to the proximity of disease in the bone itself, will account for some of the cases where paralysis has occurred during the course of an acute catarrhal otitis, or of a chronic purulent affection with caries, or necrosis. In the former, relief of tension will cure the paralysis, while in the second the occurrence of the paralysis is a sign of the serious nature of the bone lesion. Cases I and III are samples of these.

Where paralysis occurs in the course of an ordinary acute otitis media, one is apt to expect an abnormally close relationship of the

nerve to the mucous membrane of the tympanum. Concussion of the nerve over the canal at the time of operation will account for some of these instances where paralysis follows several days after the operation, although the canal itself was not exposed.

Where the lesion follows immediately upon the operation, McKernon is of the opinion that the nerve has either been injured, or partially, or completely, severed. This is, however, not always true, otherwise the speedy recovery in some cases would not have occurred.

Involvement of the nerve in scar tissue in the Fallopian canal has been reported by Alt. Relief was afforded by operation.

Pressure from a gumma in a case of Syphilis, where there is rapid loss of internal ear hearing, may produce facial paralysis.

Hemorrhage into the facial nerve in the Fallopian canal is a possible contingency.

Complete destruction of the nerve, apart from those unfortunate cases where the nerve is divided during operation, is believed to be the condition where there is a Tuberculous process present in the ear on the side affected. In Tuberculosis Milligan states that facial paralysis is a frequent and early symptom, and reports forty-five cases with proved Tuberculosis.

Loss of power in the seventh nerve has accompanied paralysis of the sixth, and may be explained as due to extension of a suppurative process along the bone between the Eustachian Tube and the Carotid canal, to the apex of the pyramid of the Petrous bone. A most instructive series of cases of this rare combination of lesions has been collected by Gradenigo, and one was reported by Barr last year.

When necrosis invades the Labyrinth, facial paralysis is not uncommon from partial destruction of the Aqueductus Fallopii, exposing the nerve to infection; but as defects in the wall may be present, the nerve lesion is not necessarily a sign of disease in the bone.

Paralysis of the facial nerve is also found associated with fracture of the base of the cranium, and here the lesion is in the neighborhood of the Geniculate Ganglion, and usually the paralysis is temporary.

That paralysis following operation is not necessarily due to injury is proved by a post-mortem on a case of Mahu's, where death resulted from a kick, and the nerve was fully examined and the canal found intact. Paralysis here was due to neuritis.

Periostitis was assigned by Gougenheim as the cause of a number of cases of facial paralysis associated with influenzal otitis.

Localizing symptoms pointing to the exact situation of the lesion may be readily appreciated by reference to what has already been mentioned, viz., that the tympanic and chorda tympanic branches arise while the facial is in the Fallopian canal. When the lesion is above the Geniculate Ganglion, taste is undisturbed; when the lesion is below the Ganglion, and above the Stapedius canal, tinnitus, slight deafness, and hyperacusis are present, probably from paralysis of the Stapedius muscle. When the lesion is below the Stapedius canal and above the root of the Chorda Tympanic nerve, there is a loss in the power of taste and an alteration in the amount of saliva secreted.

Paralysis of the face opposite to the affected ear is usually central.

Where paralysis occurs in connection with aural disease the diagnosis does not present much difficulty when the above-mentioned localizing symptoms are carefully sifted, but cases do occur where difficulties arise. It is possible that a cortical origin may exist, although a sufficient aural lesion be present. As a rule, the paralysis which is central is less intense than where it is peripheral, especially so in the temporal fibers of the nerve, and the sense of taste is unimpaired.

Nuclear lesions of the facial affect mainly the labial muscles. If there are symptoms present pointing to labyrinthine invasion—vertigo, vomiting, absence of bone conduction—the seat of the paralysis is usually in the first part of its course.

If intracranial abscess is suspected, the facial paralysis, if present, will point to a cerebellar seat for the pent up pus.

The *prognosis* in a case of facial paralysis must be always a matter of doubt, because the accurate means of differentiation are wanting, but a few observations may help us in arriving at a moderately correct estimate.

Where facial paralysis occurs in connection with an acute otitis media, speedy recovery may be usually expected upon the relief of middle ear pressure, for there is usually here a defect in the Fallopian canal.

In chronic middle ear cases, the majority of patients recover if operation be undertaken speedily, and the debris which is pressing on the nerve removed, and even if the facial nerve itself be not even looked for, except to avoid it.

In cases where the Faradic contractility has entirely disappeared in eight to ten days after onset of the facial paralysis, Bernhardt affirms that we have a severe form of paralysis, in which recovery cannot ensue under some months, and even then the recovery will frequently be incomplete. French writers maintain that if the pains at the onset are transitory, the paralysis will be brief.

When the nerve trunk has been severed by an instrument or by disease (vide Tuberculosis), recovery is usually not to be expected, even if the nerve be ligated at once, as in one case reported by Gradenigo where no recovery was apparent four months later.

The avoidance of the facial nerve in *operative procedures* about the ear, is a thought that must be ever present with the operator.

In Ossiculectomy and Curettage of the middle ear, there is great danger of wounding the nerve either through a hiatus in the bony wall of the internal ear, or by violence employed upon a naturally thin bony covering of the Fallopian canal.

In the removal of Aural Polypi there is danger of the nerve fibres being embraced in the attachment of the Polypi, and I well remember in one case the violent twitching of the face when I tightened the snare around the growth. Dench reports 92 cases of Intra Tympanic operations and apparently without any facial lesions.

In opening the mastoid cells, and in the procedure of a radical operation, however, eternal vigilance is the price of safety. Know where your nerve should be and keep away from it. Moore has well said, "Do not search for the nerve. Remove all diseased tissue—that is all." Heath claims for his operation that it is unaccompanied by liability to facial paralysis, but that can hardly be regarded as absolutely true, when we remember the relation of the nerve to the middle ear, and if I had entered the ear in Case VII, by this method, I could hardly have escaped injuring the nerve in the absence of an aditus.

I must insist upon the necessity for a good illumination of the field, and the control of hemorrhage, as necessary adjuvants to the exercise of judicious care.

Use the probe upon any doubtful point, direct the blows of the mallet, and the direction of the chisel, so as to lessen as far as possible the effect of concussion. The side of the face should be watched, of course, and for this purpose I do not cover it with a towel, but instead have it as carefully prepared as the seat of operation. Nevertheless, the main reliance must be upon incessant care in the use of the instruments.

The removal of the mastoid tip, and the opening of an abscess internal to, or below the tip, also call for the greatest care, for the nerve here may be injured after its escape from the Stylo-Mastoid foramen.

The general outline of the *treatment* may also be deduced from what has been above stated. In Acute Otitis Media, incision of the drum membrane, warm applications, purgatives—in short, relief of pressure,—will usually effect a speedy cure. This, if needful, can be followed by the Galvanic Current, massage, and strychnia internally. Burnett is emphatic in urging that electricity should not be applied until all symptoms of pain and inflammation have disappeared; but while he prefers a weak constant current applied through the mastoid fossa for three minutes every second day, McKernon advocates the interrupted current in daily doses.

In chronic ear suppuration the appearance of facial paralysis is a signal for the immediate cleaning out of the diseased bone in as thorough a manner as possible, and again it must be urged that the nerve be left alone, simply freed from pressure and left to itself.

If, however, the paralysis be of long standing, it is not to be forgotten that most brilliant results have been obtained by Alt in laying the nerve bare in the full length of its bony course and removing, it may be, scar tissue in which it has become imbedded.

Not until we have removed the diseased tissue should we attempt to transplant the paralyzed nerve into the hypo-glossal or the spinal accessory, a device which has been fairly successfully employed by Ballance, Beck, etc., in thirty-four cases.

The use of electricity in chronic cases is not so satisfactory as in acute, but it should be tried faithfully however, especially where there is evidence of the existence of a remnant of excitability.

The following *cases* have occurred in my private practice and Hospital Service:

Case I. M. H., female, aged five, was brought to my office April 27, 1897, suffering from otitis media purulenta (right) of a year's duration, accompanied by acute colds in the head. A small perforation anterior to and below the short process, some hypertrophic rhinitis, but no adenoids, were made out. After treatment the discharge ceased, and the ear remained dry for two years, when, following an attack of scarlet fever, it discharged for a few days, but I did not see the little patient. It gave no further trouble till two years later, July, 1901, when another attack developed and I found a polyp attached apparently about the junction of the pos-

terior wall and the membrane, and posterior to the original perforation, which was still present. Adenoids were now quite marked and the tonsils enlarged. These were removed under anesthetic, and the attic being found extensively carious, the ear was curetted and the malleus removed. Granulations reappeared and were removed on several occasions during the succeeding twelve months, with no concurrent symptoms, but in June, 1902, a well marked peripheral facial paralysis developed and a radical operation was performed. The mastoid was freely carious and the sinus exposed. All diseased tissue in the region of the nerve was carefully removed. A week later some betterment of the paralysis was observed, and at the end of the month, the patient was discharged showing great improvement. She was treated electrically twice a week for about two months, with some improvement. July, 1909, in a letter from her father, it is stated that there is an asymmetry in the face in smiling and laughing.

Case II. L. P., female, aged twenty-six, consulted me in September, 1900, for a chronic otitis media purulenta of the right ear, which had existed since childhood. The radical operation was performed, and nothing of special interest was observed, but on the third day, facial paralysis developed. A letter from her physician four months later, informed me that the paralysis was unaltered, and lately I learn that this patient is deceased,—but no information regarding the condition of paralysis prior, is available.

Case III. E. H. C., male, aged six, was brought to my office November 13, 1907, for a twisted face. Six weeks since the boy developed scarlatina, and ten days ago suffered from earache for two days, followed by a discharge from the left ear, which still continues, but yesterday, on opening his mouth, the jaw seemed twisted to the right. The same ear is said to have discharged for one day about three months ago. Almost complete peripheral paralysis was now present, the eye closing about half. The membrana tympani was bulging in the upper posterior quadrant, and although the canal was filled with pus, the perforation could not be brought into view. Adenoids filled the naso-pharynx. The day following, these were removed, and the membrane incised. A week later the paralysis was less marked, and owing to the rapid narrowing of the incision in the membrane, the catheter and suction were used to empty the middle ear, and argyrol injected. On the 20th of December, the discharge ceased, and on the 3rd of February the nerve had quite regained its function. Electricity was not employed.

Case IV. M. W., female, aged twenty-two, entered hospital suffering from a double suppurative otitis media of twelve years' duration, with an acute exacerbation in the left ear. The radical operation was performed on the 19th of February, 1909—antrum, aditus, and tympanum, necrotic and full of debris, sinus and dura not exposed, and the facial nerve not approached, no sign of paralysis when patient left table, but the same evening it became marked. The tip of the nose was deviated, the eye open even in sleep, lachrymation profuse, taste almost absent, mastication difficult, lodgment of the food in the cheek.

Electrical Reactions Taken by Dr. Balmer, March 18.

1. Faradism. Weak Current—No response of either nerve or muscles.

Medium Current—No response of either nerve or muscles.

Strong Current—Impossible to employ this current on account of pain produced and nervous condition of patient.

2. Galvanism. Weak Current—Contraction when applied to muscles direct. Contraction was not sluggish, but was not as brisk as normal. A weaker current produced more contraction than that of sound side. (Irritability of weakness.) A. C. C. > than K. C. C. (Reaction of degeneration.)

Medium Current—The above conditions of weak current showed more plainly.

Strong Current—Could not be used. There was no reaction when the galvanic current was applied to nerves.

At first iron and strychnia were administered. After three weeks electricity was applied, and on the 14th of April she was discharged. Improvement was first noticed about the first of April, and on the 20th of July the following notes were taken: mouth quite straight in laughing, naso-labial fold pronounced, nose straight, all eye symptoms absent except a slight sense of weakness, hyperacusis absent, senses of taste and smell still reduced in left side, left face much more sensitive than right, and power of sniffing practically absent.

Case V. E. D., female, aged sixteen, entered hospital suffering from left chronic suppurative otitis media, of eight years' standing. Operation May 12, 1909. The antrum was entered by the route of the attic, and the sinus was found unusually far forward. Both sinus and dura were exposed in removing the necrotic bone. While packing the cavity a week later, a sharp hemorrhage occurred which was controlled by packing and pressure bandage. On the seventh

day thereafter, a fortnight after the operation, the mouth was noticed to be slightly crooked, but closure of eye and wrinkling of face were perfect. Two days later, distinct paralysis was displayed by a markedly crooked mouth in laughing or whistling, and the failure to close the eye. Packing was discontinued. On the 5th of June, patient was discharged with a practically normal condition of the face. The patient reported for examination on July 15th, and the facial paralysis was completely gone.

Case VI. E. I., female, aged twenty-three, entered hospital complaining of chronic suppurative otitis media of the left ear, of six months' standing. Radical operation was performed on the 8th of June, 1909. The whole of the bone anterior to the sinus was extensively necrotic, and the sinus was uncovered. The antrum was large, and situated above the level of the tympanic cavity, with necrotic walls, and the dura was extensively laid bare. The vertical cells were implicated right to the tip. The bridge was very soft, and the facial nerve was exposed 6 mm. external to the normal situation in the vertical portion, and carefully cleaned, about 10 mm. of the nerve being visible. Paralysis was noted immediately upon recovery from the anesthetic. The Palpebral Fissure was widened, lachrymation free, failure to close the eye, wrinkles absent, paralyzed angle of mouth a trifle lower, whistling impossible, mouth crooked, senses of taste and smell not altered, no deviation of nose or of uvula and hyperacusis not present. Strychnia was administered from the beginning but no electricity was applied. Six weeks later, at time of writing, the eye could be closed slightly, the naso-labial fold was faintly marked and the face appeared somewhat more symmetrical.

Case VII. A. D., male, age seventeen, entered hospital with typhoid fever. During convalescence, symptoms of mastoid irritation appeared in the right ear, in which there had been a chronic suppurative otitis media for fourteen years, accompanied during the entire period by partial facial paralysis. On examination the palpebral fissure is widened, eye cannot close, furrow below orbit, and naso-labial fold present, brow wrinkles absent, and lachrymation noted only in cold weather. Right angle of mouth lower than left, and drawing of mouth to left side in speaking and laughing. Right ear burns and is quite red. Twitching of mouth and nose is frequent, sensation of right side of face diminished. Increased secretion of saliva is intermittent, and mastication, unless slow and deliberate, is fatiguing. Action of palate and uvula normal, sense of taste unaffected, no hyperacusis.

Dr. Balmer reported that with the Faradic current the contraction is good except in the occipito frontalis, the risorius, buccinator, and orbicularis oris, where it was present but faint.

On C. galvanic excitation the contraction was sluggish, more especially in the superior maxillary region, the K. C. C. being greater than the A. C. C. On direct stimulation of the three main branches of the facial nerve, the muscles supplying the upper jaw responded much less than those for the eye and lower jaw.

Radical operation was performed on the 21st of July, and proved to be of more than usual interest and difficulty because the bone was sclerosed throughout, the antrum was obliterated, the middle fossa very low and the shoulder of the sinus unusually far forward. I have referred to this, however, in a separate communication.

The search for the facial nerve and the location of the seat of pressure was attended therefore with unusual difficulties, but by using the semicircular canal as a guide, it was finally located and exposed at a point corresponding to the usual situation of the elbow in the floor of the aditus. At this point the Fallopian canal apparently communicated with the middle ear by a fine sinus, in which lay an extension of the thick pyogenic membrane which lined the middle ear. A further hiatus in the bony wall was located just above and posterior to the foramen ovale, occupied similarly by membrane and debris. The bridge between the sinus and hiatus was removed and the nerve trunk freed as far as prudence seemed to warrant. On account of the stress to which the nerve was necessarily exposed during these manipulations, an increase in paralysis was expected, but for the first twenty-four hours, the indications of facial paralysis were materially lessened, particularly about the mouth and eye; after that time, the original paralysis returned and became much more marked, and in addition there was present loss of taste in right half of tongue, inability to sniff or smell, and deviation of the tip of the nose to the left. At the end of the first week, at the time of writing, considerable modification exhibited itself in all of the symptoms.

For six years I have looked upon Case I as cured, but, in view of her father's letter, I must investigate the condition of the facial nerve at the earliest opportunity, and, if possible, locate the remaining lesion. It is probable that nothing can be effected now by treatment.

In Case III there was undoubtedly a hiatus in the wall of the canal, and inflammatory products caused undue pressure through the opening directly upon the nerve.

The paralysis in Case IV will probably disappear entirely, and was likely due to concussion.

The lesion in Case V was probably due to direct pressure from the packing, or to the contraction of a blood clot, the sequela of the hemorrhage—the source of which was presumed to be an injury to the sinus wall from the forceps used in packing.

In Case VI the exposed position of the nerve led to its being injured during the operative manipulations, but I look for a complete recovery. The anatomical arrangement is exceptional, but serves as an indication of the need for perpetual watchfulness while operating.

Of Case VII it is too soon to speak finally. If the expected recovery does not take place, and the electrical reactions continue to warrant it, I shall proceed to search the nerve trunk for a possible fibrous pressure point.

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PRENTICE AND DEAN: *Annals of Otology, Rhinology and Laryngology,* December, 1906.

47 Grosvenor St.

REPORT OF A COMPLICATED CASE OF ACUTE MASTOIDITIS, OF UNUSUALLY RAPID PROGRESS RESULTING FATALLY; AUTOPSY.*

BY A. SPENCER KAUFMAN, M. D., PHILADELPHIA.

The case which I wish to report is of particular interest for two reasons: first, on account of the great rapidity with which not only the surrounding structures were involved, but also organs in other parts of the body, by metastasis; secondly, it points out the great amount of damage that may be done by well-meaning relatives to relieve the patient's sufferings.

Case: W. M. S. R., age fourteen, white; schoolboy. Mother died of tuberculous peritonitis, and one sister has tuberculosis cutis. Many of the family are mouth-breathers. The patient had scarlet fever in infancy and measles at the age of four, but any ear complications with these illnesses were denied.

Six months prior to the present illness the boy had a slight earache on the right side, which promptly disappeared. This same condition was repeated four months later. About one month after this he began to have a muco-purulent nasal discharge, which continued up to the time of the present trouble. No history of a running ear can be elicited. The boy has been a mouth-breather all his life.

Two days before I saw the patient he had some slight pain in the right ear, which was gone the next morning, but the following night returned with increased severity. To relieve this pain the father dropped hot melted butter into the ear, but after the temporary relief afforded by the heat, the pain grew more intense. The next day the patient became steadily worse, developed high fever, with low, muttering delirium. There was chilliness (but no distinct rigor), anorexia and nausea. His physician was called, and he, in turn, asked me to see the patient. This was the night of February 6th.

I found the boy extremely restless and talking at random, jumping from one subject to another without coherence. He would answer questions intelligently if he was spoken to in a loud voice and the question repeated. Temperature was 101.2°F., pulse 112, and respiration 20. Tongue heavily coated, and breath foul. Skin parched and hot, thus presenting a typical picture of toxemia.

Examination of the right ear showed the auricle to be pushed far forward, the swelling and redness and tenderness extending over

*Read by invitation before the Section on Otology and Laryngology, College of Physicians of Philadelphia, April 21, 1909.

the mastoid and down the neck. The external auditory canal was found to be blocked with the butter that had been poured in the night before, and after this was removed a view of the membrana tympani could not be obtained, as the canal was so edematous that the calibre was almost entirely obliterated. Immediate operation was advised, and the patient removed to Jefferson Hospital.

On admission to the hospital, examination of the heart, lungs and abdomen was negative. Reflexes were normal. Pupils reacted normally, were regular and equal. Urine was turbid, amber in color; acid; specific gravity 1030; trace of albumen; no sugar; urea, 2.5.

First Operation, 1:00 A. M., February 7th.—As soon as the knife entered the skin at the tip of the mastoid, a quantity of greenish-yellow, foul-smelling pus spurted out. This, we found, after making the complete mastoid incision, did not come from a perforation through the cortex, but had worked its way through the soft parts from the tympanum, completely surrounded the cartilaginous canal, and extended down into the superficial muscles of the neck. The mastoid process was greatly discolored, especially at the tip. Upon removing the cortex, the cells were found to be entirely broken down and the process filled with pus and debris. A horizontal incision running backward from the first one was necessary in order to remove all the necrotic bone. There was a perisinus abscess, and the sinus itself, which was exposed through a necrotic opening about as big as a dime, was yielding, but covered with granulation tissue. There was some doubt as to the advisability of leaving the sinus intact, but, as the patient had had no rigors, and on account of so much free pus in the wound, which would in all probability infect the possibly sterile sinus, were it opened, we decided to wait, and if necessary do a second operation to evacuate the sinus if it showed signs of infection. The dura was exposed through the tegmen antri, but was found to be pulsating, of good color, and not bulging. The posterior wall of the osseous canal was removed down to the annulus tympanicus, but, as no opening through the tegmen tympani could be felt with the incus hook, the contents of the tympanic cavity were not disturbed, making a so-called Heath, or modified radical, operation. Flaps were made of the cartilaginous canal and the posterior wound partially closed, drainage being carried out both posteriorly and anteriorly.

The boy stood the operation well, and was apparently much improved, his temperature, however, remaining at about the same

point as when he was admitted. The blood count after the operation showed a leucocytosis of 29,300, with a polynuclear percentage of 69. The next day the leucocyte count dropped to 11,700, while the polynuclear percentage rose to 76. The night of February 8, or almost forty-eight hours after the first operation, he began to develop distinct rigors and run a typical sinus thrombosis temperature. A second operation was decided upon the next day.

Second Operation, February 9th.—The sutures were removed from the original wound and the sinus was now found to be very firm. The bony covering was removed for a greater distance above and below the part originally uncovered by the necrotic process. Upon opening the sinus a firm clot was removed, but no pus was found. The head was lowered and the sinus curetted in both directions, procuring free bleeding, which was allowed to continue for a few seconds, to wash out any hidden clots, and then promptly controlled by packing. Not being satisfied, in the presence of such virulent infection, to allow the tympanic cavity to remain undisturbed, the tympanic ring was now removed by means of the electric burr, and it was while doing this that a drop of pus was seen to come through the dura. The dura was incised and a pair of Jackson brain-forceps introduced through the opening, evacuating about a half-ounce of pus. This cavity was drained with iodoform gauze.

The lower angle of the wound was found to communicate with two pus cavities in the neck, one anterior and one posterior to the sternocleidomastoid muscle. These, however, did not involve any deep structures. Two counter-openings were made in the neck and these cavities drained with rubber draining tubes. The cavity of the mastoid was packed with iodoform gauze and only the angles of the wound were brought together with sutures.

After this operation, which was also well borne by the patient, he ran a sub-continued type of temperature, ranging from 101° to 104°. The leucocyte count increased to 22,000 and the polynuclear percentage dropped to 70, and he seemed to be holding his own until four days later, when his temperature suddenly shot up to 106°, and from that time on he ran a pyemic type of fever. On the next day numerous patches of dullness developed, scattered over the posterior surface of both lungs, with many rales, fine and coarse. The respiratory murmur approached the puerile type. There was another reduction in the leucocyte count to 16,600, and an increase in the polynuclear percentage to 75. The following day signs of broncho-pneumonia were more extensive, the pulse became irregular, and at times was so weak that it could not be

counted at the wrist. He lapsed into unconsciousness on the morning of February 16th, and died in the afternoon.

During the entire time the patient was in the hospital, he had no nausea or vomiting, or any other symptom of intracranial pressure, and he was singularly free from pain.

Following the second operation a culture was taken to make auto-vaccine, but, as it could not be standardized in time, a stock vaccine of 50,000,000 streptococci was injected without any signs of reaction. He also received seven intravenous injections of Collargol at the suggestion of Dr. S. Solis-Cohen, who saw the case as medical consultant. It is interesting to note that whenever the polynuclear percentage increased, indicating additional infection, there was a decrease of the hyperleucocytosis, showing lessened resistance.

Autopsy.—The points of particular interest in the post-mortem examination, as performed by Dr. John Funke, are as follows:

On the superior surface of the left ventricle of the heart there was a beginning milk spot. The left lung showed crepitus throughout; on the lower lobe, along the middle of the fissure, showed a hard, indurated area surrounded by hemorrhagic infiltration. The center of this mass was necrotic. There was an area similar to this on the base of the upper lobe. The right lung showed scattered pyemic emboli and the peribronchial lymph nodes were enlarged. The spleen was enlarged, dark red in color, and showed hyperplasia. The cortices of the kidneys were bulging. The liver was large and mottled and the under surface showed a small abscess five centimeters in diameter. On section, the liver showed fatty infiltration. Peyer's patches were swollen.

The meninges were slightly edematous, and there was some slight injection of the meningeal vessels. The right temporosphenoidal lobe, posteriorly and inferiorly, contained a circumscribed ovoidal cavity which extended into the underlying brain substance for a distance of two centimeters. The bottom of the cavity was composed of grayish-pink necrotic, granular substance. The margin of the cavity was composed of similar tissue. The remaining brain substance contained nothing unusual.

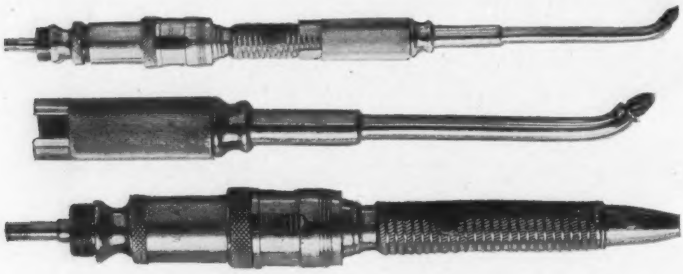
The lateral sinus of the right side was obliterated at a point where it was exposed through the mastoid process. This opening perforated the petrous portion of the temporal bone, making an opening eight centimeters in diameter and connecting with the cavity described in the brain. The sinus as it passed through the base of the skull contained a purulent grayish-white substance.

No. 1429 Spruce Street.

AN INSTRUMENT WHICH FACILITATES THE REMOVAL OF THE EXTERNAL NASAL WALL.

BY C. WESLEY BISHOP, M. D., MINNEAPOLIS, MINN.

In draining the antrum of Highmore I have often found it difficult to obtain a sufficiently large opening in the nasal wall, the various instruments at my command being in one way or another not quite adapted to the particular case in hand. This, of course, is not to be wondered at, considering how the nasal chambers vary in size and contour, so that an instrument that can be used easily in one case will be useless in another. I have designed the instrument herein described with the idea that its simplicity and flexibility would render it more adaptable to the varying demands than any instrument



I had used heretofore. A glance at the accompanying drawing will explain the construction of the instrument, which consists of three parts, viz: (a) A flexible shaft made to fit into the chuck of the handle of a Victor drill outfit. (b) A burr which is screwed into the distal end of the shaft. (c) A sleeve covering the shaft. This sleeve is bent at an angle of fifty degrees at one end, and the other end fits over the handle-piece. The instrument is inserted into the nose, the bent end down. When the burr is opposite the site of election, a slight turn brings it in apposition to the field of operation, and, the burr rotating, it is necessary to exert only slight pressure in order to make an opening of any desired size in the antral wall.

The simplicity of construction renders sterilization easy, and, as the working end of the instrument is only three-sixteenths of an inch thick, it can be used in the narrowest nose.

No. 702 Pillsbury Building.

A CASE OF PRIMARY TUBERCULOSIS OF THE FAUCES, POSTERIOR PHARYNX AND PALATAL AND LINGUAL TONSILS, CURED BY TUBERCULIN INJECTION.*

BY JOHN R. WINSLOW, B. A., M. D., BALTIMORE.

The treatment of tuberculosis of the upper air tract has ever been a bête noir of laryngology.

While some encouraging results have been obtained by the older methods, the mortality percentage has been so appalling that any real addition to our therapeutic resources will be received with rejoicing.

Tuberculous lesions rarely heal spontaneously, those involving the soft palate, uvula, tonsils, anterior pillars and structures posteriorly, being, according to Roberty Levy, of absolutely unfavorable prognosis. Lesions involving the posterior pharyngeal wall may, however, be comparatively benign.

Primary lesions are much more sluggish and less malignant than secondary ones.

The use of tuberculin preparations in selected cases of tuberculosis of the upper air tract is well warranted, but as yet there are but few recorded cases of cure, especially in this country.

The following case is reported as a small contribution, affording some encouragement in persistence along this new avenue of hope in this direful condition.

Miss L. F., a young lady of 18 years, consulted me October 30, 1907, complaining of some pain on swallowing, worse latterly, and an excess of mucous secretion in the throat (pharynx). She was of slight physique, a little pallid, but not emaciated. Temperature normal.

Her parents are living and of robust physique, but she has a brother in the latter stages of pulmonary tuberculosis. No history of heredity obtainable.

She has suffered from the present trouble for over a year, and has been treated by her family physician, who applied silver nitrate solution to the posterior pharynx, but admitted that he did not suspect the true nature of the ulceration.

Upon examination, the posterior wall of the naso-pharynx was seen to be covered with a tenacious secretion, beneath which a superficial ulceration the size of a fifty-cent piece, with indolent base,

*Read before the meeting of the Southern Section of the American Laryngological, Rhinological and Otological Society at Richmond, Va., February 12, 1909.

was discovered; on the left side this extended up behind the posterior palatine arch into the epipharynx. At its outer edge a fusiform growth resembling a lateral band was seen, behind the posterior arch. Ulceration of the rudimentary palatal tonsils, as well as of the lingual tonsils, existed but the larynx was uninvolved.

The granuloma of the posterior pharynx was removed with punch forceps and submitted to Professor J. L. Hirsch, of the University Hospital, who reported it as a typical tuberculoma (giant cells and caseation). Examination of a smear from the ulcerated surface showed a few scattered tubercle bacilli.

Careful examination of the chest at this time, and subsequently on several occasions, failed to reveal any lung involvement.

From the negative chest examinations and the absence of laryngeal lesions or symptoms during the whole period, I believe this to have been a case of primary inoculation of the pharynx, with the consumptive brother as the source of contagion.

The patient was put upon the usual systemic and hygienic treatment of tuberculosis, and being extremely fearful of the extension of the lingual ulceration to the larynx, I instituted vigorous local treatment, with mops of lactic acid, gradually increasing from 25 to 100 per cent.; the same local treatment was applied to the other ulcerations as they subsequently appeared. Orthoform lozenges were prescribed for the relief of dysphagia at meal time.

When first seen, the lesions were limited to the regions named, but within a few days extension to the adjoining structures of the fauces began.

The earliest appearances in each region were an edema and sub-mucous infiltration with minute tubercles, followed by ulceration, and often a melting away of the entire structure within a few hours. It was most interesting and at the same time disheartening to observe this identical process successively in the posterior and anterior palatine arches, the uvula and velum palati, until these structures were ultimately in great part destroyed.

Becoming alarmed at the persistent progress of the infiltration despite vigorous constitutional and local treatment, I referred the patient to my colleague, Dr. Gordon Wilson, Associate Professor of Medicine in the University of Maryland, and Director of the Municipal Tuberculosis Hospital, for examination and consultation.

After elimination by careful examination of detectible lung involvement, we agreed upon the employment of injections of tuberculin. These were instituted by Dr. Wilson on January 9th, 1908, and continued until July 31st, 1908.

The preparation used was a mixture of equal parts of Bouillon filtrate (B. F.) and tuberculin (T. R.).

Tuberculin (Denys), bouillon filtrate, "B. F."

This product consists of the filtrate from bouillon cultures of the tubercle bacillus and contains all the soluble products elaborated by the bacteria while grown on bouillon. It differs essentially from "old" tuberculin in that *no heat* is used in its preparation.

Tuberculin R, "T. R."

For the preparation of this tuberculin, bacilli from a virulent culture are dried *in vacuo* in the dark and then pulverized. The powder thus obtained is suspended in distilled water and centrifuged at high speed for about forty-five minutes. The opalescent liquid above, containing no bacilli, is decanted, and constitutes the *Tuberculin oberer,* which is discarded.

The precipitate consisting of the bacterial residue is again dried *in vacuo,* suspended in water and centrifuged; the residue obtained is treated in the same manner. This is done several times. The mixture of the fluids obtained at the subsequent operations constitutes tuberculin R. (Tuberculin Rueckstande). It is supplied in liquid form, and the solution contains 2 mg. of powder to each cc.

The *dosage* was variable, but was in the main progressively increased from the minimum of 1-10000 mgr. of each on January 9th to the maximum of 2.5 mgr. of each on July 31st. The frequency of injections was quite variable, from a seven-day interval at the beginning, to one of three to four days at the termination of treatments.

The relation of dosage to frequency can better be understood by reference to the accompanying chart.

There were no "reactions" during treatment in the ordinary sense of the term, but during February there was a slight conjunctivitis, and during May a marked one, which were undoubtedly "reactions."

Weight—January 9th (winter clothes), 102 pounds.

July 31st (summer clothes), 109 3-4 pounds.

Gain in weight, 7 3-4 pounds, plus difference in weight between summer and winter clothes.

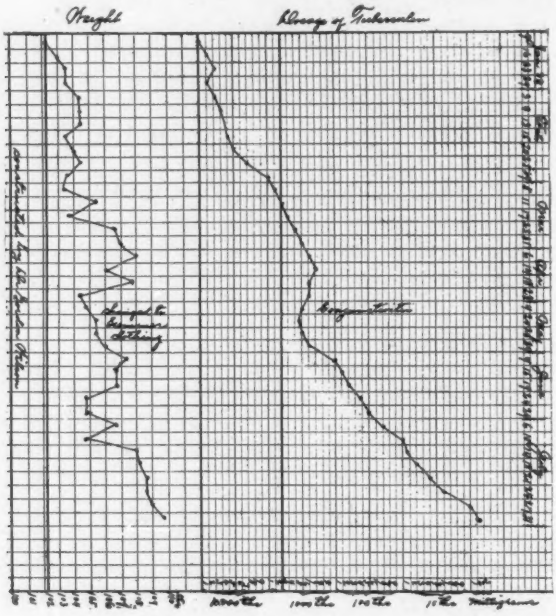
At the termination of the treatment, on July 31st, 1908, the patient was in excellent general health. Locally, the greater part of the posterior pharyngeal wall presented a smooth cicatrix; the posterior palatine arches were destroyed and the anterior ones par-

tially so; the uvula and about one-half of the velum were destroyed; the palatal and lingual tonsils were replaced by a cicatrix.

The patient was given a cleansing spray and directed to continue her general treatment.

She was re-examined by me in October last, more than a year after the first visit, and found in excellent general and local health. I learn that she has passed through the present winter without disturbance of any kind.

I am aware that the objection might be raised in this case that the cure was effected rather by the hygienic, systemic and local



treatment employed, than by the tuberculin. In reply to which I can only insist that, aside from the post-pharyngeal ulceration, there was absolutely no sign of arrest in the progress of the disease prior to the employment of that agent.

This case emphasizes two main facts:

1. The value of tuberculin in localized tubercular lesions.

2. The importance of early diagnosis, and of regarding as suspicious all abrasions and ulcerations of the mouth, fauces, tongue and pharynx, which will not heal under customary treatment.

No. 114 West Franklin Street.

THE PARS SIGMOIDALIS SINUS LATERALIS AND ITS RELATION TO PROCESSUS MASTOIDEUS OSSIS TEMPORALIS.

BY H. J. H. HOEVE, M. D., DES MOINES, IOWA.

Knowing very well from text-books, literature and actual experience that the idea exists among surgeons and anatomists, that the pars sigmoidalis sinus lateralis may be found anywhere in the processus mastoideus, occasionally even directly behind the meatus acusticus externus, I have given this subject a little close study in order to find, if it is possible to tell by examination from the outside how far the sinus in question reaches forward.

The os temporale of a full term baby can be divided into three pieces, although not quite as readily as one of a seven months fœtus. 1. A flat bent plate (*Pars squamosa*) marked by a shallow fossa (*fossa articularis mandibulae*) and a small process of bone (*processus zygomaticus*). The plate is round except at its lowest part where a small portion being lacking it is marked by a concave margin to which is attached the second portion. 2. A bony ring (*Annulus tympanicus*), the upper part of which is formed by the concave margin of the pars squamosa. 3. Pars petrosa (*Pyramis*). This portion is not yet marked by the processus mastoideus, as the development of the latter is incomplete until the second or third year after birth. The processus mastoideus varies greatly in size in different animals and human beings. At birth it is flat and corresponds to the base of the pyramis and its air cells do not appear until puberty, at which time it displays its conspicuous appearance as seen in the adult.

The position of the sulcus sigmoideus varies in infants and adults. In infants and before the development of the cellulae mastoideae, the sulcus being much nearer the surface of the pars squamosa. In the adult the pars squamosa is attached to the pars petrosa at its postero-inferior border (*Fissura petrosquamosa*) and on account of the facies superior pyramidis (the same as facies anterior pyramidis (Spalteholz) corresponding to the floor of the fossa cranii media, the topography of this fissure would correspond to the level of the latter. In the fœtus the anterior end of the fissure always corresponds to the anterior extremity of the concave defect of the pars squamosa, the location of which in the adult would be a point just above the middle root of the zygoma. In the fœtus, the posterior end of the fissure corresponds to the junction of the posterior part of the pars squamosa and the pars mastoidea, a point

(*Incisura parietalis*), into which in the adult is fitted the angulus mastoideus ossis parietalis. A line drawn in the adult between the two points mentioned corresponds to the topography of the floor of the fossa cranii media.

At the location where the fusion of the pars mastoidea and the pars squamosa takes place there is frequently a remnant of a suture present (*Sutura squamosomastoidea*) which, according to Treves, is marked in about one-third of all adult skulls. This suture in the full term babe runs downward and forward from the incisura parietalis to the posterior angle of the concavity mentioned at the lower part of the pars squamosa. In the adult it is well marked in thirty-four per cent of all cases, most frequently on the right side and, on account of the pars sigmoidalis sinus lateralis lying on the internal surface of the processus mastoideus, just in the junction between it and the facies posterior pyramidis, it must have an important relation to the topography of the sinus. In the adult I find that the postero-inferior margin of the pars squamosa overlaps the pars mastoidea in every case sufficiently to give rise to the upturned part of the cresta supramastoidea (*Linea temporalis inferior*). Consequently the latter is not always found at the same place. If a line is drawn from the incisura parietalis to the spina suprameatum, which represents in every case the natural location of the sutura squamosomastoidea, we can readily see the relation of the cresta supramastoidea (*Linea temporalis inferior*) to the former in different skulls.

The parieto-squamomastoid angle (*Incisura parietalis*) is possibly one of the most important landmarks we possess in connection with the processus mastoideus, as it gives us the location of: 1—The posterior part of the true level of the fossa cranii media; 2—The upper part of the genu of the pars sigmoidalis sinus lateralis; 3—The upper end of the sutura squamosomastoidea; 4—The point where the sinus petrosus superior empties into the pars sigmoidalis sinus lateralis.

TOPOGRAPHY OF THE FLOOR OF THE FOSSA CRANII MEDIA.

Most frequently the topography is given as corresponding to the posterior root of the zygoma or supramastoid crest (*Linea temporalis inferior*). This is correct for about one-quarter of an inch just above the meatus acusticus externus, but for the rest, as mentioned before, the linea temporalis inferior varies greatly in position and therefore I prefer to draw a line between two fixed bony points. A line from a point just above the middle root of the zygoma (*post-*

glenoid tubercle) on a level with the upper margin of the arcus zygomaticus to the incisura parietalis (A-B). In fifty-five per cent of all skulls examined, this line was found above the prominent ridge caused by the cresta supramastoidea (*Linea temporalis inferior*) and in forty-five per cent it was found below the same. In ten per cent of my skulls the incisura parietalis formed an excavation of at least 3 mm. depth in which cases it is better to draw the line to a point one quarter of an inch posterior to the incisura parietalis on the sutura parietomastoidea. In twenty skulls which could be differentiated first by their history and second by their characteristics as having belonged to right and left-handed persons, (eighty-five per cent right-handed and fifteen per cent left-handed) this line was found above the linea temporalis inferior in forty per cent of the right-handed and below it in forty-five per cent. In the left-handed skulls it was above it in ten per cent and below it in five per cent. In all the skulls the linea temporalis inferior had the same appearance on both sides of the individual skulls.

TOPOGRAPHY OF THE SINUS LATERALIS.

Of the seventeen right-handed skulls (eighty-five per cent) fifteen (eighty-eight and eight-tenths per cent) had the sinus sagittalis superior empty into the sinus lateralis dexter. One of them emptied into the sinus lateralis sinister, (the skull of a holdup man) and one emptied into both, (the skull of a young colored criminal). In the three left-handed skulls the sinus sagittalis superior emptied into the sinus lateralis sinister.

TOPOGRAPHY OF PARS HORIZONTALIS SINUS LATERALIS.

Piersol mentions a line drawn from a point (asteric 2.5 cm. above and 3.8 cm. behind the center of the meatus acusticus externus to a point 3.8 cm. above the inion, which represents the superior limit of the sulcus.

In no case in cranial surgery is it necessary to use a tape measure or any kind of an instrument to measure with, as there are sufficient natural landmarks. It is sufficient to know the exact location of the pars horizontalis sinus lateralis in order to avoid it with the trephine or chisel. If in nearly all right-handed skulls, the sinus sagittalis superior passes into the sinus lateralis dexter, then in order to map out the sinus lateralis it must suffice for the surgeon to draw a line from the incisura parietalis to a point one-half of an inch to the right of the inion, as the confluens sinuum lies mostly a little to the right of the inion. The sinus lateralis may be a little above or below this line, but the incisura parietalis always marks the most su-

perior level of the genu of the sinus. For those surgeons who are not familiar with the contents of the cranium and its anomalies, possibly Birmingham's rule is a good one. He states that: The region of danger in trephining is enclosed by two lines, one from a point 3.3 cm. above and 3.8 cm. behind the center of the meatus acusticus externus to a point 12 mm. above the inion, the other from a point 3.8 cm. behind the meatus acusticus externus and on the same level, to a point 12 mm. below the inion. He states that the sinus almost never passes beyond these limits in either a downward or upward direction.

TOPOGRAPHY OF PARS SIGMOIDALIS SINUS LATERALIS.

The topography of this part of the sinus lateralis varies greatly and it is this portion of the sinus which especially has drawn my attention. Mecewen gives us the direction of the sinus by stating: A line drawn from the upper edge of the meatus acusticus externus to the asterion and another one from the parietosquamomastoid junction to the tip of the processus mastoideus indicates the direction of the sinuses, the latter corresponding to the midportion of the sinus. The latter line mentioned is undoubtedly the best one to use for studying the relations of the pars sigmoidalis sinus lateralis as it represents the topography of the angle formed at the external part of the posterior surface of the pyramis, by the junction of the latter with the internal surface of the pars mastoidea ossis temporalis.

In the series of twenty skulls differentiated as to having belonged to right and left handed persons, seventeen were right-handed, and of these seventeen in only one was the sinus sagittalis superior found to open into the sinus lateralis sinister, and that was the skull of a hold-up man, and in another one it opened into both sinuses, and that was the skull of a young criminal. In the three left-handed skulls the sinus opened into the sinus lateralis sinister.

Whenever the sinus sagittalis superior opens into the sinus lateralis dexter, the latter is of greater size than its fellow of the opposite side, and correspondingly forms a deeper groove in the bones and at the place where its course is altered forms deeper indentations. This is possibly the reason for the pars sigmoidalis sinus lateralis extending farther forward on the right side, as is generally stated in text-books, but which is not true in every case. It stands to reason that in right-handed people the left cerebral hemisphere does most of the intellectual work and consequently has a greater cellular activity than the right, and it is also plain that this must be the cause

of the difference in size between the two cerebral hemispheres. In right-handed people the left is always a little larger than the right, except under pathological conditions, which either are inherited or which were present very early, as we shall see later on. If the left hemisphere is larger than the right we can expect that the upper part of the hemisphere presses the least fixed point of the falx cerebri over to the opposite side to a greater or lesser extent and that seems to be sufficient cause for the sinus sagittalis superior to empty into the sinus lateralis dexter, as it simply has to follow gravity. That this theory is correct is shown by the skull of the hold-up man, the one right-handed skull in which the sinus sagittalis superior emptied into the sinus lateralis sinister, and the genu sinus sigmoidalis came forward just as far on the left as on the right side, for the right temporal region was abnormally large in this case, there being at least a difference of 8 mm. between the distance from the medial sagittal plane. It seems that this pathological condition of the right cerebral hemisphere is the cause for the sinus sagittalis superior in this case emptying into the sinus lateralis sinister.

DIFFERENCES BETWEEN THE SINUS LATERALIS DEXTER ET SINISTER.

The right sinus came from 5-15 mm. farther forward than the left in six out of all the right-handed skulls, and from 1-5 mm. in five cases and in two cases it was on the same plane as on the left. In four of the right-handed the left sinus came 3 mm. farther forward than the right. Of the three left-handed cases the sinus lateralis sinister came from 5-8 mm. farther forward in two cases and 3 mm. farther than the right in one case. These measurements were taken from the spina suprameatum and tallied with the ones taken from the incisura parietalis.

THICKNESS OF BONE BETWEEN THE PARS SIGMOIDALIS SINUS LATERALIS AND THE OUTER SURFACE OF THE PROCESSUS MASTOIDEUS.

In sixteen out of seventeen right-handed cases the bone was thicker on the left than on the right side, varying from 2-8 mm. In the left-handed skulls the right side was thicker, varying from 1-6 mm.

EXTERNAL SIGNS OF TOPOGRAPHY OF SINUS.

Having inspected all the skulls closely, I find that only eight out of the seventeen right-handed skulls are more prominent over the left part of the occiput, seven were equally prominent on both sides and two slightly more prominent on the right side. Of the left-handed ones two were more prominent over the left side of the

occiput and one was equally prominent on both sides. A study of the differences in the distance from the medial sagittal plane to the tip of the processus mastoideus, to the most prominent part of the processus mastoideus and to the incisura parietalis, etc., did not reveal anything new, except that it brought out the fact that in right-handed people the left distances are frequently greater.

THE LINEA SQUAMOSOMASTOIDEA AND ITS RELATION TO THE GENU SINUS SIGMOIDALIS.

In considering all the skulls, I find that the upper level of the genu sinus sigmoidalis corresponds in nearly every case to the upper one-fourth of a line drawn from the incisura parietalis to the spina suprameatum (*Linea squamosomastoidea*). Frequently it corresponds to the posterior one-half of this line, especially on the right side.

RELATIONS OF THE SINUS TO THE TRIANGLE FORMED.

The genu sinus sigmoidalis never passes above the linea squamosomastoidea, but always comes up to it at its upper part, especially on the right side. A line drawn from the incisura parietalis to a point just above the middle root of the zygoma and another line from the incisura parietalis to the tip of the processus mastoideus form two sides of a triangle, the base of which is partly formed by the meatus acusticus externus. The linea squamosomastoidea divides it into two parts, the posterior part being the one on which the topography of the part sigmoidalis sinus lateralis should be outlined. It is safe to say that the lower quarter of the entire processus mastoideus is not in relation to the sinus and is separated from it by the digastric fossa (*Incisura mastoidea*), but it should be borne in mind that this groove is occasionally very shallow. On the right side the topographical line frequently comes forward to the middle of the posterior part of the triangle, and on the left side it frequently remains behind the triangle altogether in right-handed people. The anterior angle of the triangle corresponds to the trigonum suprameatum, the superior angle to: 1, the incisura parietalis; 2, the posterior part of the true level of the fossa cranii media; 3, the upper part of the genu sinus sigmoidalis; 4, the upper end of the sutura squamosomastoidea; 5, the point where the sinus petrosus superior empties into the pars sigmoidalis sinus lateralis. The inferior angle corresponds to a relatively safe area, or at least the safest area where the surgeon may start to open the cellulae mastoideae when such a procedure is necessary. A line drawn from the posterior end of the incisura mastoidea across the lower end of the

processus mastoideus to the lower level of the meatus acusticus externus will mark off this inferior angle, A place which is not quite as safe as the one just mentioned lies just behind the posterior border of the meatus, and it can readily be seen from the accompanying cut, that the antero-inferior part of the processus mastoideus is safer than the rest.

SUMMARY.

1. The natural topography of the fossa cranii media is a line extending from the incisura parietalis to a point just above the middle root of the processus zygomaticus ossis frontalis (*post glenoid tubercle*).

2. A line drawn from the incisura parietalis to the spina suprameatum gives the natural location of the sutura squamosomastoidea in every case, above which the sinus never extends.

3. The incisura parietalis corresponding to the parietosquamomastoid angle corresponds to the location of: (1) the posterior part of the true level of the floor of the fossa cranii media; (2) the upper part of the genu sinus sigmoidalis; (3) the upper end of the sutura squamosomastoidea; (4) the place where the sinus petrosus superior empties into the sinus sigmoidalis.

4. In over half of all cases the floor of the fossa cranii media lies higher than the linea temporalis inferior.

5. In fifteen out of seventeen right-handed people (98.2 per cent.) the sinus sagittalis superior empties into the sinus lateralis dexter. In all left-handed people three left-handed of a series of twenty the sinus sagittalis superior empties into the sinus lateralis sinister.

6. A line extending from the incisura parietalis to the tip of the processus mastoideus represents the topography of the angle formed between the posterior surface of the pyramis and the inner surface of the pars mastoidea.

7. The cause of the sinus sagittalis superior emptying into the sinus lateralis dexter in right-handed people is the greater size of the left cerebral hemisphere.

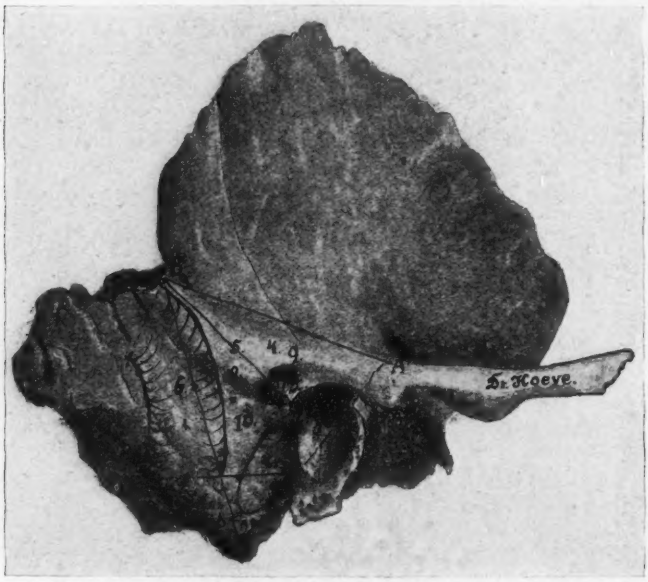
8. In 35.2 per cent. of all right-handed people the pars sigmoidalis sinus lateralis extends from 5-15 mm. farther forward on the right side; in 29.4 per cent., 1-5 mm., and in 11.7 per cent. it extends forward just as far as the left, and in 23.5 per cent. the left extends 2 mm. farther forward than the right.

9. In 66.6 per cent. of all left-handed people the pars sigmoidalis sinus lateralis extends from 5-8 mm. farther forward on the left side, and in 33.3 per cent. 3 mm.

10. In 94.1 per cent. of all right-handed people the bone between the pars sigmoidalis sinus lateralis and the outer surface of the processus mastoideus is from 2-8 mm. thicker on the left side. In left-handed people the right side is from 1-6 mm. thicker in all cases.

11. The upper level of the genu sinus sigmoidalis corresponds in every case to the upper quarter of the linea sqaumosomastoidea.

12. As no one is able to look inside of the processus mastoideus and know the exact position of the pars sigmoidalis sinus lateralis, it is the safest to start chiseling at the antero-inferior part of the same.



THE TOPOGRAPHY OF THE PARS SIGMOIDALIS SINUS LATERALIS.

1. Middle root of the processus zygomaticus (*postglenoid tubercle*). 2. Spina suprameatum and fossa suprameatum. 3. Incisura parietalis. 4. Cresta supramastoidea (*Linea temporalis inferior*). 5. Linea squamosomastoidea. 6. Pars sigmoidalis sinus lateralis. 7. Safest place to start chiseling for mastoid operation. 8. Fossa mastoidea. 9. Anterior part of the triangle described. 10. Posterior part of the triangle described. A, B, C, the entire triangle.

K. of P. Building, Room 230.

DIRECT LARYNGOSCOPY IN THE DIAGNOSIS AND TREATMENT OF PAPILLOMATA OF THE LARYNX.

BY RICHARD H. JOHNSTON, M. D., BALTIMORE.

Before the introduction of direct laryngoscopy it is probable that some cases of papillomata of the larynx in children escaped diagnosis. In children with hoarseness as the only symptom, there was formerly no satisfactory method of finding the cause. In some cases examination with the mirror was possible, but in many patients the diagnosis was guessed at. The direct laryngoscope has changed what was formerly speculation into certainty. There is no longer any excuse for laryngologists to remain in ignorance of the pathological condition in infants' and children's larynges. The examination of the smallest larynx is as easy as the mirror method in adults. When it became possible to examine the larynx in the extended position we realized that a decided step forward had been made, though the cramped position of the operator and the tense muscles of the patient left much to be desired. When Mosher described the "left lateral position" it seemed impossible because the average man does not understand how one can see around a corner through a straight tube. I was sceptical about the flexed position until I succeeded in getting a better view of the child's larynx than I had ever gotten in the extended position. One is able to see around the corner because the tisues in a child are so yielding and there is no difficulty in pulling the base of the tongue and the epiglottis upward. With Jackson's modified slide speculum the best possible view of the child's larynx is obtained for diagnosis and treatment. To illustrate the ease and efficiency of the flexed position I will refer to two cases of papillomata of the larynx which I have seen during the past year.

K. E., four years old, was seen at St. Joseph's Hospital in April, 1908. For a year she had been hoarse and for some months dyspnoeic. When she entered the hospital symptoms were such that a foreign body in the larynx was thought of. This probable diagnosis was strengtened by the radiograph which gave a distinct shadow in the larynx. With the patient anesthetized, unsuccessful attempts to see the larynx with the mirror were made. I was asked by Drs. Carroll and Duker to try direct laryngoscopy. At that time I knew nothing of the flexed position, so the ex-

amination was made under chloroform in the extended position. We made a positive diagnosis of papillomata and attempted to remove the growths. The child promptly ceased to breathe and a tracheotomy had to be done. At a later date the larynx was examined in the flexed position and the papillomatous masses cleaned out with ease. From time to time this procedure has been repeated, with the result that at this writing the patient breathes through the larynx for hours. The tendency of papillomata to recur is well known. I believe the flexed position gives us the best method of watching and treating these growths. The patient can be examined as often as desired without anesthesia by holding securely the legs, arms and head. I have made the examination in children from fourteen months to six years of age without anesthesia and have never failed to get a satisfactory view of the larynx.

The second patient, aged three and one-half years, was brought to the Presbyterian Hospital a month ago. She had had increasing dyspnoea for several months. The child was placed on the table, the head flexed on the chest, Jackson's modified speculum introduced, the base of the tongue and the epiglottis pulled up and the diagnosis of papillomata quickly made. All present saw the growths. No anesthetic was used, the legs, arms and head being held by assistants. The next day the patient was given chloroform for tracheotomy and died on the table from paralysis of the heart. It was our intention to remove the growths through the direct laryngoscope. In a patient, aged twenty-two years, it was found impossible to remove an anterior papilloma by the mirror method. She was anesthetized and the growth successfully removed through the speculum in the flexed position. When one has tried both extended and flexed positions he will never use extension in children again. In flexion the operator stands in a comfortable position or sits if he prefers it and uses the speculum in the left hand, having the right hand free for operative procedures. In stenosis of the larynx from other causes, the location and extent of the pathological lesion is quickly diagnosed; it can be seen at a glance what treatment will be best for the individual case. In two cases of stenosis examined in the flexed position it was seen that the lesion was anterior and that the opening posterior was large enough for small intubation tubes which were immediately introduced. Direct laryngoscopy in extension is one of the triumphs of modern medicine; flexion is far superior to extension in children, and we are indebted to Mosher for giving us such a simple method

of examining the child's larynx. The time is not far distant when all laryngologists will have to be as expert in direct laryngoscopy as with the mirror if they wish to keep in the front rank of our specialty. Since the procedure has become so simple it is within the reach of all and anyone can master it if he is willing to give the proper amount of time to it.

919 N. Charles St.

AN UNUSUAL CASE OF CHRONIC ANTRAL SUPPURATION.*

BY WILLIAM R. BUTT, M. D., PHILADELPHIA.

Patient was a colored woman, fifty-four years of age. She gives a definite history of syphilitic infection when she was fifteen years of age, and says that at that time she had severe ulceration of her throat. The only treatment she received was some throat wash.

She says she had no trouble with her nose and could always breathe well through it, until she was twenty-six years of age. Then she had trouble with both tear ducts (nasal ducts) and a swelling appeared between the side of her nose and her right eye. This swelling was "lanced" and eventually disappeared. About this time also, an ulcer, which began on the right side, formed a large perforation of the septum. There was much crusting on both sides of the nose. A little later both nostrils became occluded at about the same time, and they have remained so ever since. She has never been able to smell anything since that time, but she has some sense of taste.

Examination.—Outside of nose and face negative. (The left eye has been removed and the left frontal sinus opened seven years ago as the result of an accident; but this seems not to have any connection with the condition being reported.) There is a purulent condition of both tear ducts.

*From Dr. George B. Wood's service at the Howard Hospital.

Examination of Nose.—With a speculum, all that could be seen in either nostril was a smooth hard wall, vertically placed, blocking the lumen of the fossae about three-quarters of an inch inside the vestibules. This mass seemed to run directly into the septum on one side and appeared on the other in about the same position. The walls of both nostrils in front of this were smooth and no turbinates were visible. Both nasal fossae were the same, except that on the right a probe could be passed along the floor of the nose under the obstructing mass a short distance, but not into the pharynx.

Examination of Mouth and Throat.—The naso pharyngeal orifice was contracted by scar tissue to the diameter of a lead pencil and would not admit the finger. The left side of the hard palate, opposite the bicuspids and molars was bulging and fluctuating.

Treatment—Patient refused to enter the Hospital for a thorough operation under ether, so that, after injecting cocaine, a trocar and canula was pushed into the mass in the left nasal fossa. It passed through a thin, bony partition into a large cavity, and a large quantity (at least 2 ounces) of stinking greenish-yellow pus was evacuated. The opening was enlarged as widely as possible with a sphenoid sinus punch. The cavity occupied the region of the left nasal fossa, antrum and ethmoid cells. A probe could not be passed from this cavity into the pharynx nor into the mass in the other nasal fossa. By pressing on the swelling on the hard palate pus was forced out through the opening made in the nose; and this swelling on the hard palate, which was in the soft tissues, disappeared at once. Patient refused, at the second and last visit, to have the other side (the right) operated on. She was much improved and said she was going back South. There were no symptoms referred to the right side (unless the dacryocytitis was due to obstruction of the nasal duct).

The most probable explanation of this case seemed to be that disease of the left antrum had forced the outer wall of the left nasal fossa inward, through the perforation in the septum, as far as the outer wall of the right fossa. The fact that the masses in the two fossae did not communicate at the time of the operation might be attributed to a constriction of the mass where it passed through the septum (into a sort of hour-glass shape) and that this constriction had afterward closed up.

1701 Chestnut St.

ON THE TREATMENT OF CICATRICIAL STENOSIS OF THE LARYNX BY THE METHODS OF O'DWYER AND ROGERS.*

BY D. BRYSON DELAVAN, M. D., NEW YORK.

In the successful management of stricture of the larynx, three things are absolutely essential.

1. That the stricture be relieved.

2. That the relief be permanent.

3. That the treatment of the case be conducted with the least possible injury to the parts involved.

Many operators have endeavored to treat these cases, and by various means. Some, like Sir Morell Mackenzie, Whistler, Schroetter and Navratil have used intralaryngeal methods. Others have resorted to external operations of a more or less extensive character. Treatment by occasional and temporary dilatation has almost invariably proved a failure, while operative measures have generally left the case worse off than it was before. It remained for O'Dwyer to solve the problems of the situation and to effect a method by which all of the necessary indications were completely met and the disadvantages of other methods avoided. First, he proved the hitherto unknown fact that the larynx would tolerate the presence of a foreign body for indefinite lengths of time. Then he devised the O'Dwyer tube, an instrument which, with suitable modification, fulfills in a remarkable degree the indications present in the cases under our discussion.

No great contribution to surgery has ever been more modestly presented, nor has surgery often received a gift more indicative of important future results than the article published by O'Dwyer in the *Medical Record* for June 5, 1886, entitled "Chronic Stenosis of the Larynx Treated by a New Method, with Report of a Case." How thoroughly the author himself appreciated its value will appear from his own words: "Chronic Stenosis of the larynx is one of the most unsatisfactory diseases which the physician is called upon to treat. Although I have treated only a single case of it with my laryngeal tubes, I am fully convinced that they will prove infinitely superior to anything yet devised for the relief of this unfortunate class of sufferers."

*Read before the Section of Laryngology of the British Medical Association at the annual meeting in Belfast, July 23-31, 1909.

Two years after the foregoing was published, O'Dwyer, in the following remarkable statement, says: "Had intubation of the larynx proved a complete failure in the treatment of croups, I would still feel amply repaid for the time and expense consumed in developing it, for I believe that it offers the most rational and practical method yet devised for the dilatation of chronic stricture of the glottis." *N. Y. Medical Journal,* March 10, 1888.

Since the publication of O'Dwyer's prophetic article, the value of his method has been recognized and has been applied with success by large numbers of experimenters in many parts of the world. Many who at first objected to it are now its firm advocates. Moreover, an ever-increasing amount of clinical evidence based upon the actual treatment of cases has been accumulated, all of which tends to prove that intubation, used under proper conditions, has succeeded in effectively meeting the indications present in the treatment of chronic stenosis of the larynx.

The work so ably begun by O'Dwyer has been taken up by others, notably by Dr. John Rogers, Jr., of New York, through whose keen appreciation of the possibilities, rare technical skill and untiring ingenuity and patience, the method of O'Dwyer has been brought to a high degree of perfection, and it has been used with success not only upon adults, but upon children and even upon very young infants.

The method of intubation may be employed without tracheotomy or with tracheotomy, either as simple intubation or associated with fixation of the tube through the fistula.

Among the other varieties of laryngeal obstruction to which it is applicable, those which come within the limits of our discussion or which represent conditions which may give rise to the formation of cicatrices are as follows:

I. Cicatrices of traumatic, operative or inflammatory origin. When following tracheotomy the deformities may be due to: (a) Contraction of the posterior soft part of the trachea following separation anteriorly by the canula of the tracheal rings; (b) Dislocation of the cricoid forward and downward, especially when the fistula is through or close to it; (c) Overriding of the cut anterior ends of the tracheal cartilages in too long a fistula; (d) Inversion of the cut anterior ends of the tracheal rings, especially if the fistula is to one side of the median line; (e) Contraction of the trachea round the canula, due to sloughing of the cartilages or rings; (f) Spur formation posteriorly, especially in combination

with contraction of the posterior soft portion of the trachea, or dislocation of the lower segment forward and the upper backward, or both.

II. Granulations round a canula or intubation tube, which are much less common than is generally believed.

III. Hypertrophic subglottic laryngitis, most commonly encountered in retained tube cases.

IV. Prolonged and repeated diphtheritic inflammation, which must be exceedingly rare in these days of antitoxin.

Before attempting to describe the O'Dwyer-Rogers method, a word must be said as to the principles upon which it is based and the indications which justify its use. It is essential that these indications be very clearly understood. The interior of the larynx is practically a cavity or tube having rigid inelastic walls and lined with various soft tissues, the latter liable to injury and therefore to the formation of cicatrices. The presence of cicatrices at the posterior aspect of the larynx is likely to interfere with the normal physiological movements of the dilators of the glottis as well as to encroach upon the glottic space. Anteriorly, a lesion situated at the apex of the vocal bands is analagous to one located in any other angle of the body, as, for instance, between the fingers or at the corner of the mouth. A solution of continuity of tissue in any such angle is sure to result in adhesions as the process of healing advances and, in many cases, such adhesion if left to itself, will only be limited by the extent of the pre-existing lesion.

The history of scar tissue is much the same wherever cicatrices may be found, for example, any attempt to divide web fingers without closing the wound at the apex of the angle in such a manner that the granulating parts cannot come together will surely result in failure and will produce a condition of adhesion worse than that which existed before. This is one of the oldest recognized surgical facts.

Nor is it only in the case of V-shaped wounds that this tendency to adhesion is observed. In nasal surgery, it is a matter of common occurrence that unless injured parts have been kept separate from each other until healing has been fully accomplished, adhesion is likely to take place. The same is true of the larynx, in which the incision of a thin web of tissue may possibly be successfully accomplished, but incisions into dense scar tissue will almost invariably re-unite, even after long-continued dilatation.

The incessant activity of the movements of the larynx and the delicacy of its soft parts render it peculiarly liable to accident and to the conditions which we are considering. None of the plastic methods possible in other situations have been made available in the larynx without resort to laryngo-fissure.

What has just been said at once suggests the fact also that the attempted removal of cicatrices from the larynx is not likely to be attended with ultimate success, since it appears that in most cases, with the lapse of a relatively short space of time, the intra-laryngeal obstruction has reappeared. This has been demonstrated time and again in another connection, namely, in the attempt to relieve dyspnœa, due to abductor paralysis of the larynx, or other causes by which the vocal bands have fallen together to such an extent that proper respiration was impossible. In such cases the performance of laryngo-fissure and the removal of one or both vocal bands has almost invariably resulted in ultimate failure. So also when the attempt has been made to excise cicatrices, or to incise them, and afterwards dilate, ultimate failure has been in even greater measure the rule.

It cannot be too clearly understood that *operations upon the larynx, whether from within or without, tend in themselves to the production of stricture.* In view of these facts it would seem necessary that in order to obtain permanent success, the cure of this condition should be based upon principles different from those involved either in simple temporary dilatation, incision of the scar with dilatation or with resection of soft parts, as has heretofore been practiced.

The principle of treatment upon which the greatest success in these cases seems to depend is, that scar tissue in general, when subjected to long continued stretching, will finally lose its resiliency. For this there are many illustrations; as for example, the stretching of the cicatrix in the case of a scar following a laparotomy, or following an operation for hernia and the like. Instances are not wanting to prove that the same results have been obtained in the larynx. Proof of this would seem to lie in the fact that cases of cicatricial stenosis in which the stricture has persistently returned after relatively short periods of intubation have been cured by long continued retention of the tube.

In order to succeed in producing satisfactory pressure upon the cicatrix, two things are absolutely necessary:

First, that the instrument be adjusted with great accuracy to the shape as well as to the size of the stricture. The ordinary O'Dwyer

tube will not answer the purpose. Each case must have its own tube which must be modified in every instance to meet accurately and completely the special requirements of the conditions in hand. To adapt the tube properly to the case will demand on the part of the operator much skill and experience and a practical development of the sense of form and proportion. All of these qualities and attainments are possessed by Dr. Rogers in a high degree and it is by reason of them that he has been enabled to bring the method to its present perfection.

Second: That the intubation instrument used be large enough to exert steady and active pressure force upon the contracted tissues, stretching them up to the limit of possibility. Moreover, in order to insure permanent success, the stricture must be under tension for a considerable length of time. A year, in some cases possibly much longer, may be required.*

In the words of Dr. Rogers himself: (*American Journal Med., Sciences*, April, 1908)

"The principle which I have found successful in the treatment of hypertrophic laryngitis and in cicatrices of the upper part of the respiratory tract involves only long continued and constant dilatation, which stretches the contracted tissues to the utmost limit they will bear without sloughing and up to the largest normal caliber of the passage in question, and so overcomes the obstruction by producing a permanent pressure atrophy in the affected parts.

To accomplish the continued dilatation and consequent pressure-atrophy necessary to overcome the obstruction of either a hypertrophic laryngitis or a cicatrix, a properly made O'Dwyer intubation tube is almost a perfect instrument. It can be worn without harm to the patient for months or years, and I have observed such patients with the tubes in place to pass successfully through measles, pneumonia, typhoid fever, and even whooping-cough; indeed, it does not seem appreciably to increase the gravity of the prognosis of these affections. The tube, as is well known, possesses a head which rests on and in the aryteno-epiglottic folds, a neck constricted from side to side, to fit between the vocal cords within the glottis, and below a retaining swell which expands to occupy the space at and below the cricoid cartilage. The lower end in the children's sizes is made longer than in those used for adults, as it was designed to extend well beyond the false membranes of a laryngeal diphtheria. The tip is again constricted

*For a full description of the treatment of Chronic Stenosis of the Larynx by Intubation, see Rogers, American Journal of the Medical Sciences, November, 1905, and April, 1908.

from side to side to pass easily through the glottis. The "Normal" tubes were designed by Dr. O'Dwyer from casts and measurements to fit somewhat loosely the average larynx and exert the least possible pressure upon acutely inflamed tissues compatible with retention of the instrument. When this stage of the disease has passed and been succeeded by a chronic thickening or a cicatrix, the tube, to obtain the pressure necessary to produce an atrophy of the abnormal thickening, must be modified into a "special" instrument for each case. That is, with the patient under deep general anaesthesia, a number of normal tubes are passed through the larynx until one is found in which the retaining swell distends the constriction to the limit the operator believes it will bear without sloughing. If the difficulty is dependent solely upon a soft hypertrophy, the diameter of the retaining swell will be regulated largely by the diameter of the passage within the cricoid cartilage. Inasmuch, however, as the retaining swell lies when the tube is in place just below this point, where the trachea is capable of considerable distension without damage, quite a little force in the test-tubage is permissible. Having thus ascertained the largest possible diameter of the larynx a "special" tube is made of the length of the normal tube suitable for the age and development of the patient but with a retaining swell having the same diameter as that of the trial tube. The neck of this "special" tube, however, to exert pressure upon the hypertrophied intralaryngeal tissues, should be as large as possible, but its transverse diameter must be 3/32 of an inch smaller than the retaining swell, or it will certainly be coughed out. Also the head must have a diameter 6/32 of an inch greater than the retaining swell to prevent the instrument from slipping within the larynx. Those measurements apply to the ordinary hard rubber tube, but when the heavier metal tube is employed the diameter of the head should exceed that of the retaining swell by 7/32 or even 8/32 of an inch, especially for adults. For very small children these measurements can be changed from thirty-seconds to sixty-fourths of an inch. It must be borne in mind that a difference of 3/32 of an inch in the transverse diameters of the neck and retaining swell is the very least which experience has demonstrated is sufficient to keep the tube in place, and many patients will require a tube with the neck 4/32 or even 5/32 of an inch less in diameter than the retaining swell. Only a trial will show what is necessary. But the neck of the special tube must be considerably larger than that of the normal, or a failure to cure will probably result.

When auto-extubation complicates the difficulties, and it may be a very dangerous complication if assistance is not constantly and immediately available, tracheotomy is almost always necessary. Some patients with hypertrophic laryngitis will cough up any tube which can be inserted, and unless it is quickly replaced, will strangle. Under these circumstances it is generally essential to provide an opening in the trachea, though there can be no expectation of thus accomplishing a cure of the stenosis. Indeed, it will probably only add a cicatrix or a deformity in the shape of an obstructing "spur" in the posterior wall of the trachea if the cannula is worn long enough. The operation should be performed carefully in the median line below the cricoid cartilage, under full anaesthesia, with the tube in place. At the same time opportunity is thus given to obtain the necessary measurements for making what I have described as a "plugged" or "clamped tube." This is a "special" intubation tube with the lower end (if there is a cicatrix low down) enlarged to exert pressure upon the stenosed area. The "clamp" or "plug" portion of the tube is a removable attachment designed to be fastened to the tube at right angles through the tracheal fistula after the tube is inserted, and thus prevent its displacement. The larger tube will support a "plug" or a cylindrical piece of hard rubber, which is long enough so that when in place its outer end extends through the fistula about an inch beyond the surface of the neck, to allow easy manipulation, and its inner end, perforated to correspond to the lumen of the intubation tube, screws into a hole in the anterior wall of the intubation tube. The "clamp" is employed for smaller instruments and is made like a pair of obstetrical forceps, of two little silver blades, which are passed through the fistula into grooves on the sides of the tube and held there by a "collar" screwed down around the shanks.

Of these two attachments the plug is probably better for large or for metal tubes, and the clamp for smaller instruments and those made of rubber. The clamp is also better in that it can be made small enough not to press apart appreciably the severed anterior segments of the trachea, and thus, by preventing contraction in the soft posterior wall, there will not be much danger of narrowing the caliber of the passage when the instrument is removed. A tube with either of these attachments cannot be coughed out and can be worn for an indefinite period without harm, and in all respects is infinitely better than the ordinary cannula. If, therefore, a patient with a hypertrophic or cicatricial stenosis of the up-

per air passages has to be tracheotomized, one of these instruments should be inserted as soon afterward as it can be obtained, and continued in place as advised for the usual special tube. Neither a plugged nor a clamped tube should, however, be employed unless there is some good reason for it, as the tracheal fistula gradually, of course, becomes permanent, and presents its own difficulties, although these may be offset by the ease with which after removal of the tube any adductor spasm can be met.

The usual intubation tube is made of vulcanized rubber, which is an ideal material, as I have never known it to clog and it is practically indestructible. It does, however, have to be removed and cleaned about once in a month or six weeks, as the head generally becomes foul; and this decomposing matter might cause sepsis or possibly obstruct the lumen. But occasionally in cases of hypertrophic laryngitis, the special rubber instrument, even after months of continuous use, will seem to produce no improvement. Then by changing to a similarly made metal tube, the desired result will generally be obtained. I am unable to explain this except that the metal tube is heavier and so may exert more pressure; or there may be some electrolytic effect, as it is constructed of bronze or brass heavily plated with gold and so consists of at least two metals which are in contact with moisture. Whatever reason may be given, the metal tube will often succeed when the rubber has seemed to fail, but the latter should always be tried first, as in the average case it is sufficient and safer. The metal tube soon becomes rough with a presumably calcareous deposit, which catches the secretions, and in at least one of my cases thus produced asphyxia. After wearing such an instrument, preferably of hard rubber, for months, it is very necessary to consider the adductor spasm both for diagnosis and treatment. When a cure ought to have been obtained the conditions may seem entirely unchanged, and the true state of affairs can only be ascertained by general anaesthesia. Under ether then, about once in every three or four months the tube should be carefully removed and the respiration observed. If it is still completely or partially obstructed, the dilatation must be resumed. But if the breathing is natural and easy, then spasm may be confidently diagnosticated, especially if in the first effort at coughing, as consciousness comes back, the dyspnoea occurs. To overcome this there must be inserted for several days or weeks a tube with a head and retaining swell and length like the previously worn dilating instrument, but with a neck as small as possible, in order that motion to some extent can occur in the

openers of the larynx. If the dyspnœa makes its appearance slowly during the course of hours or days, it is not due to adductor spasm and there is no escape from the previous treatment. When enough time, or some one or two months have passed with the very small necked tube in the larynx, and the faulty muscles may be considered to have regained their strength and the patient his power to innervate them, the small tube is removed under ether and a little morphine is administered to allay spasm and nervousness. For the first few hours, however, the attendant and the patient will suffer much anxiety, as frequent attacks of stridulous inspiration and more or less dyspnœa will occur during the readjustment to new conditions. But with patience and the assistance of antispasmodics a day or two will suffice to overcome all such difficulties.

A fibrous stricture is managed in exactly the same way, with the exception of the primary dilatation in case the stenosis is too tight to admit a tube. Under these conditions there is already a tracheal fistula, or one is urgently needed, and through this, under general anæsthesia, urethral sounds are passed up and down until considerable resistance is encountered. Then measurements are taken upon an ordinary tube and a "special" one is constructed having its specially enlarged part placed to correspond with the location of the stricture and with regard to the normal diameter of the air passage, which is capable of much stretching without damage except within the cricoid ring. When the latter has been cut in a hasty previous operation there seems to be little danger of sloughing under almost any reasonable pressure, but if it is intact, such an accident is conceivable and actually occurred in a case cited by Dr. O'Dwyer. Sometimes in very dense strictures a small tube can be inserted for a week or two and afterward a larger size. But no stricture should be considered hopeless provided any trace of mucous membrane remains, as I have had experience in at least one such case in which the passage through the larynx had to be opened by a careful dissection very much like an impermeable stricture of the urethra. A small tube was then inserted and the wound partially closed, and later larger instruments were employed, and eventually an almost perfect result was obtained.

As regards the prognosis and duration of treatment in patients with hypertrophic laryngitis or cicatricial stenosis, the statistics of the twenty-three cases previously reported furnish much information. One case of hypertrophic laryngitis (Case I), the only one in the

series which seemed to have occurred spontaneously, and without a previous diphtheria, recovered permanently at the end of about four weeks of dilating tubage. Another of cicatricial stenosis (Case XXIII., of Dr. Simpson's, previously reported as a failure) required forty-nine months, or a little over four years, of continuous intubation, *and during the latter two of these four years, the tube was not once removed from the larynx.* It has now been out for over six months, and there is no sign of recontraction and the voice is returning. Cases X, XII and XIII, the most discouraging of the series of instances of hypertrophic laryngitis complicated by cicatrices due to tracheotomies and explorations through the thyroid cartilage, required respectively about six, three and five years of dilating tubage to effect a cure. This may now be considered permanent, as the patients have remained for nearly two years without any sign of difficulty and all have good voices. Cases X and XIII would have recovered sooner if I had had less impatience and more experience, for as soon as they seemed well their tubes were removed, with the result that the cicatrices recontracted. A fibrous stricture apparently requires at least three years of continuous dilatation to the utmost limit of the normal lumen of the respiratory tract before it can be considered as permanently overcome. Even then it may gradually reappear, and if it does, another six months or a year of continuous tubage must be endured, but there is a reasonable certainty, as proved by experience, of thus gaining an ultimate cure. Cases of hypertrophic laryngitis are slow and troublesome, but once well do not recur like a cicatrix. Case XII., one of hypertrophy, had to wear a dilating tube from February, 1904, until March, 1906, and then, owing to adductor spasm, a small tube until the following June, and thus required a little over two years of treatment, which seems to be about the average."

The possibility of the successful application of intubation in the chronic laryngeal stricture of infants has been abundantly proved through the brilliant series of cases operated upon and reported by Dr. Rogers. While a majority of these were due to hypertrophic laryngitis, some were complicated with cicatrices, and all of them demonstrated the practicability of the method.

Ten of Rogers' cases were under six years of age. There was one case each at the ages of one, two, five and six years, respectively, and three each at three and four. One case is interesting because

the stenosis was due to hypertrophic laryngitis following intubation for diphtheria, and this complicated by cicatrices resulting from several laryngo-fissures and tracheotomies, the former done for the purpose of allowing the incision and the removal by dissection of numerous cicatricial bands. At the end of two years of treatment the patient was apparently cured.

As to the merits, absolute or relative, of the procedures under discussion with us, time is an all important factor in making possible a decision. No method now in use has been under observation long enough to prove that a condition of stenosis of the larynx apparently cured by it may not at some time return. Such is the history of other strictures and such the result of experience in other cases observed, of this class.

Some interesting questions arise as to whether attempts at treatment made according to the various methods now in use may not in themselves lead to future troubles. In other words, will cases which have been treated for stenosis show at subsequent periods of the patient's life a tendency towards any special form of accident? More especially, will they or not be likely to suffer in undue proportion from degenerative changes in such tissues as may remain to them in the interior of the larynx?

The lapse of at least a number of years will be necessary to demonstrate in a given case the attainment of permanent cure and the relative immunity from undesirable remote effects. Even then the only approach to certainty will be gained from the study of considerable numbers of cases up to the very end of life.

The management of these cases by any of our present methods is trying and more or less dangerous, difficult enough for both patient and physician to cause every one of us to heartily welcome any new suggestion giving promise of better things.

1 East Thirty-third St.

PRESENT STATUS OF ORAL SURGERY.

BY HENRY GLOVER LANGWORTHY, M. D., DUBUQUE, IOWA.

The object of this paper is to create a more extended discussion on the part of rhinologists in the widening field of oral surgery, and to point out the fact that we are somewhat in danger of being outclassed by many of our dental colleagues who are beginning to specialize along this line. The time has come when every ear, nose and throat physician must make himself proficient in stomatology and allied branches or drop behind in the race. This will naturally be harder on many of our beloved preceptors of the "old school" but we younger men with time, teaching, and more experience have unlimited possibilities before us. Oral surgery, and especially its details, has been neglected by the medical profession and there would seem a need of closer study and wider experience in many of these borderland problems of medicine, surgery and dentistry. Although oral surgery may be defined as the practice of surgery about the mouth, we are prone to overlook the fact that the sterilization of tooth roots, incising abscesses, repairing decayed teeth, applying an orthodontia apparatus, etc., are surgical though minor surgical operations. The repeated assertion of the modern dentist that whenever he repairs a decayed tooth or lances an abscess, he performs a surgical operation just as much as any minor surgical operation performed by the general practitioner, is a just one. Therefore he should be accorded his rightful position in an important department of the healing art. The difference in the two surgeons, if there be a difference, is humorously expressed by Luckie as follows: "One is done by the dentist in the mouth, is somewhat difficult to perform, consumes more time and the fee is less. While the physician would receive from $5.00 to $10.00 for opening and dressing abscesses, the dentist would not."

The specialist has prided himself on the fact that generally speaking, when he mentions oral surgery, he refers to a class of operations far beyond the ability of the average dentist to perform. This ground, however, owing to the advent of the distinct and highly trained oral dental surgeon is beginning to slip from under our feet. Unless a more intimate knowledge of dental pathology is obtained, we will all be at a decided disadvantage in the diagnosis

and treatment of diseases of the mouth, and can only expect that in the not far distant future the dental-surgeon will be the one most competent to perform work in connection with the jaws. That there is tentative ground for some such belief on the part of the dental profession, on the plea that a properly trained dental surgeon would be more apt to take into consideration the true value of the teeth to the individual and avoid external incisions about the mouth and face no matter how difficult the oral operation might prove, must be reluctantly admitted. Certainly the dental profession is no longer afraid of undertaking oral surgery or looking upon it as something unusual. It recognizes the fact that time, special instruments, mechanical skill and close study nowhere bring their reward so quickly as in dealing with pathological conditions about the jaws. Leading dental-surgeons have gone to extremes in the matter of time and money in the treatment of carious teeth, abscesses about unextracted tooth roots, acute and chronic osteo-myelitis, maxillary fracture, hare lip and cleft palate. It seems incredible in this enlightened age that two of the most important phases of medicine and surgery, namely the necessity of skilled orthodontia work in children and the crying need for systematic oral prophylaxis in the entire human race, is constantly overlooked by specialists as well as general practitioners. Much of the apathy in connection with this subject has of course been due to the fact that few physicians have had the training necessary to cover so great a scope satisfactorily even though they may have possessed the skill. Fortunately these conditions are no longer true as there are now many workers along the highway all striving earnestly to bring order out of chaos, borrowing from other departments whenever necessary and slowly but surely developing the field. The claim that the medical profession has abandoned the care of the mouth and teeth to dentists, is true to a large extent at the present time but it should not be so. Most physicians have paid little attention to the teeth and mouth more, however, as in the writer's case, from ignorance, rather than disinclination. Granting that mechanical skill is so frequently of importance in the handling of fractured jaws and in the substitutions of artificial devices for tissue lost by diesase or operation, that of itself should not be a sufficient reason for turning the care of the mouth over to others, or concluding that the practitioner of dentistry with special training would have any advantage, or in fact as much, as the specialist with a post-graduate dental degree. That a large part of the work of this region must be combined is without

cavil. In dental schools an attempt is made to give students two courses on oral surgery including one lecture a week and in certain instances a high class operative clinic, as well, which is more than can be said of many medical institutions. These courses embrace not only instruction in the general principles of surgery but also their practical application to pathological conditions occurring about the mouth and face, include special attention to the difficulties encountered in cases of malposed and impacted teeth, surgical treatment of facial blemishes, alveolar abscess, necrosis of the maxillae, fractures of the jaw with devices and methods of fixing and retaining fractured and displaced bones in position, antral empyema, diagnosis and removal of tumors with microscopical examination, exsection of nerves in the surgical treatment of persistent neuralgias, cleft palate, hare-lip and dozens of other similar and unexpected operations. Somewhat companion courses in orthodontia which we physicians but dimly understand, teach not simply the technique for obtaining accurate models of the teeth and alveolar process, or the construction and adaptation of the various appliances to be used in the correction of irregularities of the teeth, but take up most seriously the association between malocclusion and nasal disturbances and such general matters as the development of the jaw and their relation to normal conditions of health. The newer features of X-ray plates for the determination of the presence of teeth that have failed to erupt at the normal time and for ascertaining the forms of roots abnormally placed, are being beautifully developed by dentists in cases of special difficulty. General surgery through the introduction of anesthesia, already owes much to the dental profession and there exists at the present time considerable more which can be utilized with benefit to ourselves and patients. It is not asking too much to expect that in the future every specialist will have some knowledge of such conditions as inflammation or suppuration of the dental pulp, diagnosis and treatment of apical pericementitis, of conditions leading to the formation of acute and chronic alveolar abscess and diseases of the peridental membranes (pyorrhea alveolaris). In large dental clinics embracing as some do almost several hundred patients daily, dental students have the opportunity of seeing a good deal that has been denied the physician. It is unnecessary however, to continue such familiar observations. Although it may be said that pathological processes attacking the jaws are in actual numbers comparatively few, the variations in clinical types seem almost endless and often prove confusing. In reality every

case becomes a law unto itself. At present, from the standpoint of the special worker, the following conditions represent most of the affections confronting the oral surgeon:

1. Removal of necrosed bone from upper and lower jaws.

2. Treatment of fractures of the lower jaw and wounds in general about the mouth.

3. Repair of hare lip and cleft palate.

4. Incision and curettement of jaw cysts.

5. Excision of tumors such as fibroma, sarcoma, endothelioma and angio and osteo-sarcoma.

6. Removal of adenoid and tonsils preceding oral prophylaxis, widening palate or any necessary orthodontia work.

7. Correction of nasal obstructions from whatever cause.

8. Treatment of disease of the maxillary sinus (empyema of the antrum of Highmore).

9. Treatment of defective jaw closure or cicatricial contractions due to burns.

10. Supervision and management of trifacial neuralgia from both medical and surgical standpoints.

11. Removal of moles or nevi about the face and lips.

Few, if any, of the subjects enumerated above will encounter the slightest objection on the part of either medical or dental professions. There are probably not many men who can cover the entire field equally well without additional study. The field formidable as it is, however, is within the grasp of the average specialist. Although the proper treatment, for instance of any and every nasal obstruction as contracted with the complex task of management of tri-facial neuralgia, would appear to be at opposite ends in a consideration of affections of the head, they ought nevertheless to be included.

In conclusion it may be stated for our own individual training at least, that too much stress cannot be laid upon the importance of any subject which deals with the teeth and jaws.

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BOOK REVIEWS.

Diseases of the Ear.

By Edward Bradford Dench, Ph. B., M. D., Professor of Diseases of the Ear in the University and Bellevue Hospital Medical College; Aural Surgeon, New York Eye and Ear Infirmary; Consulting Otologist to St. Luke's Hospital, etc. Fourth edition. Cloth. Pp. 718, with 19 plates and 158 illustrations. Price $5.00 net. New York and London: Appleton & Co.

In presenting the fourth edition of this valuable American classic the author has made such changes in the text as have been rendered necessary by the advances made in otology during the past six years.

The chapters on cerebral and cerebellar diseases have been practically re-written, giving not only the author's experience, but also giving the results of numerous other observers, in this manner the subject of brain abscess has been presented very clearly from a clinical point of view; the operative technique has been carefully considered and the results obtained by the various methods of operation have been thoroughly analyzed. Labyrinthine suppuration is given much prominence and in this chapter the recent investigations of Barany and others have been embodied; the operative treatment of labyrinthine suppuration and the technique of the operation are described in detail.

Several new plates from dissections by the author have been added, one being the completed mastoid operation, another the completed radical operation, and a third the operation for acute suppuration of the labyrinth. A new plate on craniocerebral topography has also been added, bringing the question of cerebral localization more prominently to notice.

In the opinion of the reviewer this work stands pre-eminently as the first classic in otology in the English language. M. A. G.

Diseases of the Nose, Throat and Ear.

By Charles Huntoon Knight, A. M., M. D., Professor of Laryngology, Cornell University Medical College, and W. Sohier Bryant, A. M., M. D., Consulting Otologist, Manhattan State Hospital. Second Edition. Cloth. Pp. 609, with 239 illustrations. Price, $4.50 net. Philadelphia: P. Blakiston's Son & Co., 1909.

The second edition of Dr. Knight's serviceable and practical volume appears considerably enlarged, with a section on Diseases of the Ear added by Dr. W. S. Bryant. The chapters on deviated septum and sinus diseases are more thoroughly reviewed, and the newer methods described.

Under the heading of Foreign Bodies in the Larynx, tracheoscopy and bronchoscopy are considered briefly, and the reader is referred to Dr. Chevalier Jackson's excellent work for manipulation details. Dr. Knight does not believe that the older methods of exploration will be supplanted as a matter of routine. The same opinion applies to the Hay's pharyngoscope, though the latter may be of service in difficult cases. Dr. Knight treats his subjects in a lucid, practical manner, and his conclusions are based upon ripe experience.

Eighty-five pages are devoted to the anatomy, physiology, sound-perception, physiology of equilibrium and protective mechanism of the ear. Fifty-five plates are given to illustrate these subjects. The internal ear is given due attention, and diseases of its various parts are taken up individually.

Under the subject of major aural surgery, Dr. Bryant describes the operative technic of mastoid disease (acute and chronic), sinus and jugular involvement and labyrinthine suppuration. Infectious Encephalitis, therapeutics and instruments, together with procedures and appliances are given separate chapters.

The text is printed in clear, easily readable type, and the illustrations (both original and borrowed) are unusually distinct.

The book is worthy of the attention of all those interested in these diseases. M. D. L.